# NIPER JEE Pharmacy Subjects

## Sample Paper – 7

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

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

## Instructions

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

## Part A: Pharmaceutics

**Q1.** Silver chromate $Ag_2CrO_4$ dissociates as $Ag_2CrO_4 \rightleftharpoons 2\,Ag^+ + CrO_4^{2-}$. If its molar solubility in water is $s$, the correct expression for its solubility product $K_{sp}$ is:

(A) $s^2$

(B) $s^3$

(C) $4s^3$

(D) $27s^4$

**Q2.** A drug has an oil/water partition coefficient $P = 1000$. Its $\log P$ value (base 10), a common measure of lipophilicity, is:

(A) 3

(B) 2

(C) 10

(D) 1

**Q3.** An emulsifier blend is made of 40% Tween 80 (HLB $= 15$) and 60% Span 80 (HLB $= 4.3$). The HLB of the blend (weight-fraction average) is approximately:

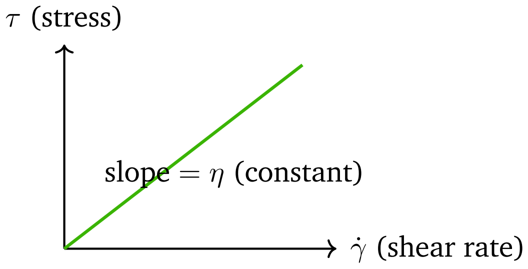
(A) 9.7

(B) 8.6

(C) 10.3

(D) 4.3

**Q4.** The flow curve below (shear stress $\tau$ versus shear rate $\dot{\gamma}$) is a straight line through the origin, so the ratio $\tau/\dot{\gamma}$ is constant at every point. A liquid obeying this is termed a:



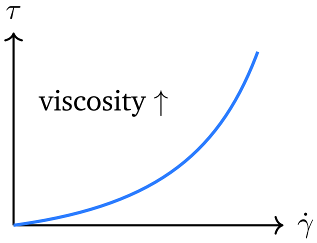
(A) Newtonian liquid

(B) pseudoplastic liquid

(C) dilatant liquid

(D) thixotropic liquid

**Q5.** The single flow curve below bows toward the shear-rate axis: the apparent viscosity *rises* as the shear rate increases (e.g. a concentrated starch paste). This behaviour is called:



(A) Newtonian flow

(B) pseudoplastic flow

(C) dilatant (shear-thickening) flow

(D) plastic flow with a yield value

**Q6.** Kinematic viscosity is defined as the dynamic viscosity divided by the density of the fluid. Its CGS unit is the stoke (St), which equals:

(A) 1 poise

(B) 1 $\text{cm}^2/\text{s}$

(C) 1 Pa·s

(D) 1 dyne/cm

**Q7.** A granulation has a bulk density of 0.50 g/mL and a tapped density of 0.625 g/mL. Carr's compressibility index of this powder is:

(A) 20%

(B) 25%

(C) 12.5%

(D) 16%

**Q8.** The angle of repose of a powder is measured by the fixed-funnel method and found to be 28°. According to the usual flowability scale, this powder has:

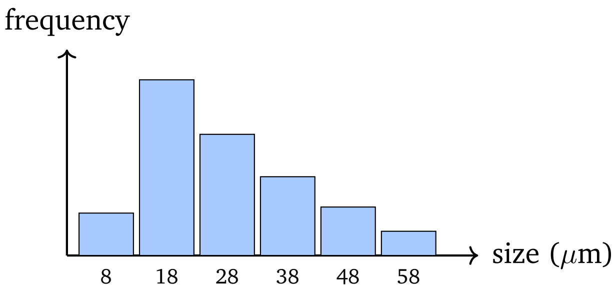
(A) very poor flow

(B) passable flow

(C) poor flow

(D) excellent (good) flow

**Q9.** The frequency histogram below shows a number–size distribution of a micronised powder. The modal (most frequent) size class, in $\mu$m, is:



(A) 8 – 18 (lower than the mode)

(B) 18 – 28 (contains the mode)

(C) 28 – 38

(D) 48 – 58

**Q10.** A buffer is made from a weak acid (pK$_a$ $= 6.10$) and its salt in a salt:acid molar ratio of 1:10. Using the Henderson–Hasselbalch equation (log 10 $=$ 1), the pH of this buffer is:

(A) 6.10

(B) 7.10

(C) 5.10

(D) 4.10

**Q11.** The spreading coefficient $S$ of a liquid over a surface is given by $S = \gamma_S - (\gamma_L + \gamma_{SL})$. A liquid will spontaneously spread over the surface when:

(A) $S$ is positive (zero or greater)

(B) $S$ is negative

(C) the interfacial tension $\gamma_{SL}$ is zero only

(D) the surface tension of the liquid is maximal

**Q12.** For two 1:1 drug–ligand complexes, complex X has a stability (formation) constant $K_X = 10^4$ and complex Y has $K_Y = 10^2$. Which statement is correct?

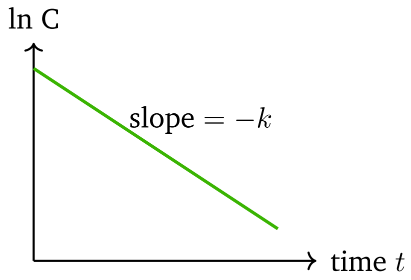
(A) Y is the more stable complex

(B) both complexes are equally stable

(C) the smaller K always means the stronger complex

(D) X is the more stable complex (higher K means greater association)

**Q13.** In the plot below, the natural logarithm of the remaining drug concentration (ln C) falls linearly with time. The degradation therefore follows:



(A) zero-order kinetics

(B) first-order kinetics

(C) second-order kinetics

(D) third-order kinetics

**Q14.** A suspension degrades by zero-order kinetics with rate constant $k_0 = 0.2$ mg mL$^{-1}$ day$^{-1}$ from an initial concentration of 40 mg/mL. The time for a 10% loss of potency ($t_{90}$) is:

(A) 4 days

(B) 40 days

(C) 20 days

(D) 200 days

**Q15.** The $Q_{10}$ of a degradation reaction is the factor by which the reaction rate changes for a 10°C rise in temperature. If $Q_{10} = 3$, then raising the storage temperature by 20°C will increase the degradation rate approximately:

(A) 3-fold

(B) 6-fold

(C) 30-fold

(D) 9-fold

**Q16.** The Langmuir adsorption isotherm is written $\frac{x}{m} = \frac{abC}{1+bC}$. A straight line is obtained when one plots:

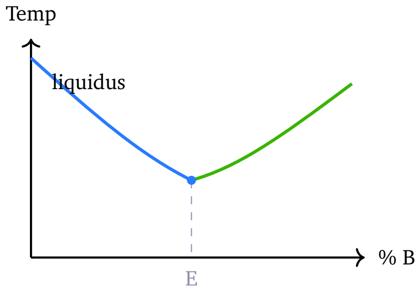
(A) $C/(x/m)$ versus $C$

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

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

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

**Q17.** The temperature–composition diagram below for a mixture of two solids shows two liquidus branches meeting at the lowest point E, where the mixture melts at a single minimum temperature. Point E is the:



(A) peritectic point

(B) inversion temperature

(C) eutectic point

(D) critical solution point

**Q18.** According to the Noyes–Whitney equation $dC/dt = \frac{DA}{h}(C_s - C)$, micronising a poorly soluble drug accelerates dissolution chiefly because it:

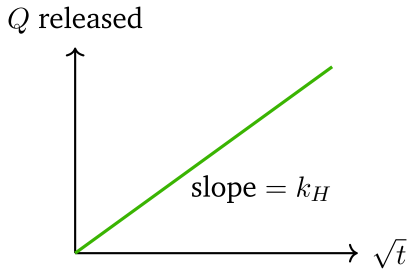
(A) raises the saturation solubility $C_s$

(B) increases the effective surface area $A$

(C) increases the diffusion-layer thickness $h$

(D) lowers the diffusion coefficient $D$

**Q19.** For a matrix-type transdermal patch, the cumulative amount of drug released $Q$ plotted against $\sqrt{t}$ gives a straight line through the origin (figure). This release pattern obeys the:



(A) zero-order model

(B) first-order model

(C) Hixson–Crowell model

(D) Higuchi (square-root-of-time) model

**Q20.** In the Korsmeyer–Peppas model ($M_t/M_\infty = kt^n$) for drug release from a *spherical* matrix, a release exponent of $n \leq 0.43$ indicates the dominant mechanism is:

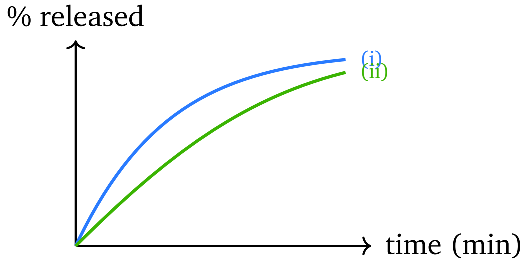
(A) Fickian (diffusion-controlled) release

(B) Case-II (zero-order) transport

(C) super Case-II transport

(D) anomalous (non-Fickian) transport

**Q21.** The two dissolution profiles below (cumulative % released versus time) compare a capsule (i) and a tablet (ii). The USP Apparatus 1, used routinely for capsules that tend to float, is the:



(A) reciprocating cylinder

(B) paddle apparatus

(C) rotating basket

(D) flow-through cell

**Q22.** An orally administered drug shows extensive first-pass (presystemic) metabolism in the liver. The most direct consequence for the same dose is:

(A) its absolute bioavailability becomes greater than 1

(B) its oral bioavailability (F) is reduced

(C) it shows zero hepatic clearance

(D) its rate of renal excretion is unchanged but absorption rises

**Q23.** Starch paste is incorporated into a wet-granulation tablet formulation chiefly to act as a:

(A) binder (adhesive)

(B) lubricant

(C) glidant

(D) film-coating polymer

**Q24.** For uncoated tablets of average weight 500 mg, the IP/USP weight-variation limit is $\pm 5\%$. The acceptable individual-tablet weight range (mg) is therefore:

(A) 490 – 510

(B) 475 – 525

(C) 450 – 550

(D) 495 – 505

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

(A) 000

(B) 0

(C) 5

(D) 1

**Q26.** A drug has a displacement value of 1.5 in cocoa butter. To prepare 12 suppositories, each from a 2 g mould and each containing 0.3 g of the drug, the total mass of cocoa butter (g) required is:

(A) 24.0

(B) 20.4

(C) 23.6

(D) 21.6

**Q27.** Anhydrous wool fat (lanolin) is able to take up considerable amounts of water yet remains greasy. It is therefore classified as which type of ointment base?

(A) absorption base

(B) oleaginous (hydrocarbon) base

(C) water-soluble (PEG) base

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

**Q28.** In a suspension, the final sediment occupies 12 mL of an original total suspension volume of 30 mL after settling. The sedimentation volume $F$ is:

(A) 0.60
(B) 0.40
(C) 2.50
(D) 0.12

**Q29.** In an o/w emulsion, the dispersed oil droplets rise and concentrate as a layer at the top, but can be redispersed by gentle shaking without the phases separating. This reversible instability is termed:

(A) cracking (breaking)
(B) phase inversion
(C) creaming
(D) Ostwald ripening only

**Q30.** The standard reference cycle for moist-heat (steam) sterilisation of aqueous parenteral solutions in an autoclave is:

(A) 160°C for 2 hours (dry heat)
(B) 121°C for 15 minutes at 15 psi
(C) 180°C for 30 minutes
(D) 100°C for 30 minutes

**Q31.** Ideally, an ophthalmic solution should be made isotonic with lacrimal fluid, equivalent in tonicity to a sodium chloride solution of concentration about:

(A) 0.9% w/v
(B) 5.0% w/v
(C) 0.09% w/v
(D) 2.5% w/v

**Q32.** The crushing strength (hardness) of a compressed tablet, expressed in kilograms-force or newtons, is most appropriately measured using a:

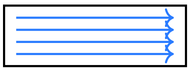
(A) Roche friabilator

(B) Monsanto / Pfizer-type hardness tester

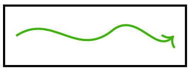
(C) disintegration test apparatus

(D) Andreasen pipette

**Q33.** The two sketches below show pipe flow: pattern (i) has smooth parallel streamlines (laminar) and pattern (ii) shows chaotic eddies (turbulent). The *transitional* region between fully laminar and fully turbulent flow corresponds to a Reynolds number range of about:



(i) laminar



(ii) turbulent

(A) less than 100

(B) exactly 1.0

(C) about 2100 – 4000

(D) greater than 100000

**Q34.** Niosomes, an alternative vesicular drug-delivery carrier to liposomes, are formed mainly from:

(A) phospholipid bilayers identical to cell membranes

(B) solid lipid nanoparticles with a crystalline core

(C) cross-linked albumin microspheres

(D) non-ionic surfactants (with cholesterol) forming bilayer vesicles

## Part B: Pharmacology & Toxicology

**Q35.** A drug is administered as a 750 mg IV bolus and the plasma concentration extrapolated back to $t = 0$ in a one-compartment model is 30 mg/L. The apparent volume of distribution ($V_d$) of this drug is:

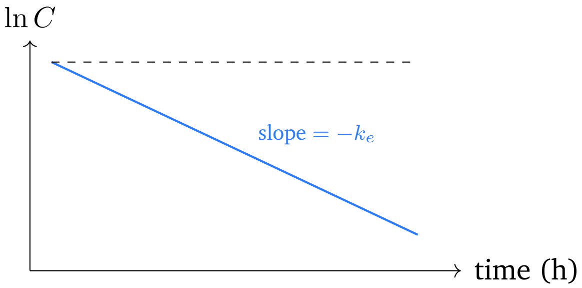
(A) 0.04 L

(B) 22.5 L

(C) 25 L

(D) 250 L

**Q36.** The semi-log plot below ($\ln C$ versus time) is a straight line, the signature of a single first-order elimination process. If the slope of this line equals $-0.0578\,\text{h}^{-1}$, the elimination half-life is closest to:



(A) 6 h

(B) 12 h

(C) 24 h

(D) 0.69 h

**Q37.** For a particular drug the area under the plasma concentration-time curve after a 50 mg oral dose is 18 mg·h/L, while the AUC after the same 50 mg given intravenously is 45 mg·h/L. The absolute oral bioavailability ($F$) is:

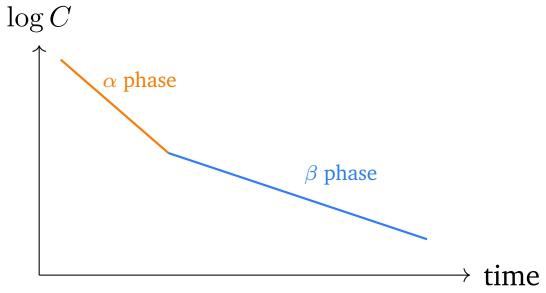
(A) 0.40 (40%)

(B) 0.18 (18%)

(C) 0.45 (45%)

(D) 2.5 (250%)

**Q38.** The semi-log plasma concentration-time curve below, recorded after a rapid IV bolus, shows a steep early segment followed by a shallower terminal segment. This biphasic profile is the hallmark of:



(A) Strict zero-order elimination throughout

(B) Flip-flop kinetics after oral dosing

(C) Saturable Michaelis-Menten metabolism

(D) A two-compartment model (rapid distribution phase then slower elimination phase)

**Q39.** A weakly acidic drug (p$K_a$ 3.5) is largely un-ionised in the acidic gastric lumen but becomes increasingly ionised as it enters the more alkaline plasma. With respect to passive diffusion across membranes, this pH-partition behaviour means the drug is:

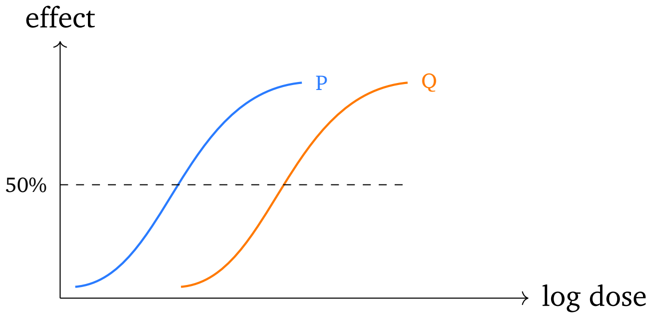
(A) Absorbed only by active transport, independent of pH

(B) Best absorbed in its un-ionised (lipid-soluble) form, and "ion-trapped" on the side where it is more ionised

(C) More readily absorbed when fully ionised

(D) Unable to cross any membrane regardless of ionisation

**Q40.** The two graded log dose-response curves below belong to drugs P and Q acting at the same receptor. Both attain the same plateau, but curve P lies entirely to the left of curve Q. The correct interpretation is:



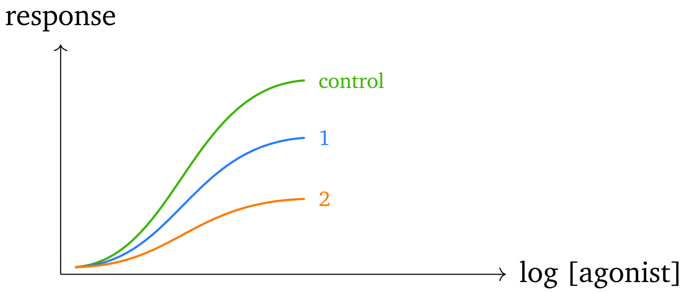
(A) P has greater efficacy than Q

(B) Q is more potent than P

(C) P is more potent than Q, while both have equal efficacy

(D) P is a partial agonist and Q a full agonist

**Q41.** An agonist is tested alone (control) and then in the presence of increasing concentrations of antagonist R (curves 1, 2). The maximal response falls progressively and is NOT restored by adding more agonist, as shown. Antagonist R is best classified as:



(A) A non-competitive (insurmountable) antagonist

(B) A competitive (surmountable) antagonist

(C) A partial agonist

(D) A chemical antagonist

**Q42.** In classical receptor theory the "intrinsic activity" (efficacy) of a drug is best described as:

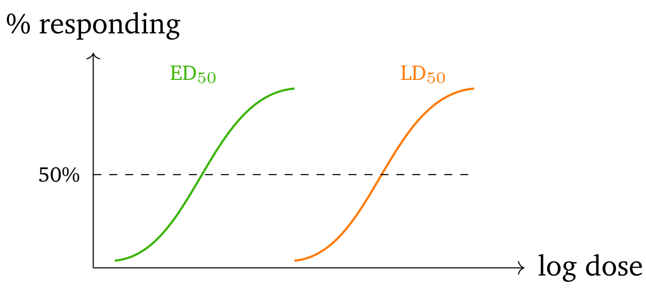
(A) The tendency of the drug to bind the receptor, measured by its dissociation constant

(B) The ability of a bound drug to activate the receptor and produce a response (1 for a full agonist, 0 for a pure antagonist, between 0 and 1 for a partial agonist)

(C) The dose required to produce 50% of the maximal effect

(D) The negative logarithm of the antagonist concentration giving a dose ratio of 2

**Q43.** The quantal dose-response curves below give the median effective dose and the median lethal dose of a drug, with $ED_{50} = 5$ mg and $LD_{50} = 200$ mg. The therapeutic index of this drug is:



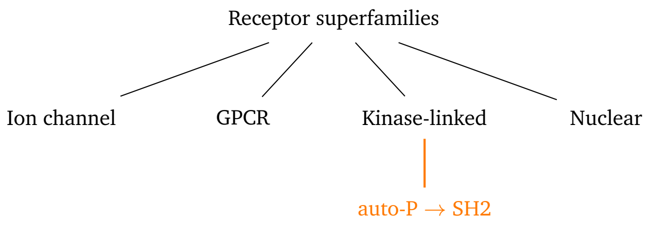
(A) 5

(B) 0.025

(C) 195

(D) 40

**Q44.** In the receptor superfamily tree below, the highlighted branch ends in "autophosphorylation of tyrosine residues → SH2-domain signalling". A physiological ligand that signals through this branch is:



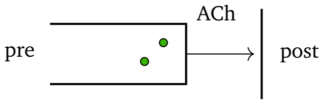
(A) Acetylcholine at the nicotinic receptor

(B) Adrenaline at the $\beta_2$-receptor

(C) Insulin (and epidermal growth factor) acting on a receptor tyrosine kinase

(D) Cortisol at the glucocorticoid receptor

**Q45.** At the cholinergic synapse depicted, echothiophate (an organophosphate) inhibits acetylcholinesterase. Compared with carbamate inhibitors such as neostigmine, echothiophate is distinguished because it:



(A) Acts only for a few minutes and is rapidly reversible

(B) Phosphorylates the enzyme to form a very stable (effectively irreversible) complex that can “age”, requiring pralidoxime for early reactivation

(C) Does not affect acetylcholinesterase at all

(D) Is a competitive antagonist at the nicotinic receptor

**Q46.** Tropicamide is chosen over atropine as a mydriatic for routine fundoscopy because tropicamide:

(A) Stimulates muscarinic receptors to dilate the pupil

(B) Is an irreversible antimuscarinic with a very long action

(C) Is a short-acting antimuscarinic, producing mydriasis and cycloplegia that wear off within hours rather than days

(D) Causes intense miosis useful for examination

**Q47.** Esmolol is favoured for controlling heart rate during intra-operative tachycardia and hypertensive emergencies because it:

(A) Is a $\beta_1$-selective blocker with an ester linkage hydrolysed by red-cell esterases, giving an ultra-short half-life of a few minutes

(B) Is a non-selective $\beta$-blocker with a half-life of many hours

(C) Acts as a $\beta_2$-selective agonist

(D) Irreversibly alkylates the $\beta$-receptor

**Q48.** Cisatracurium is a useful non-depolarising neuromuscular blocker in patients with both hepatic and renal failure because it:

(A) Is a depolarising agonist hydrolysed by pseudocholinesterase

(B) Is excreted entirely unchanged by the kidney

(C) Depends on biliary excretion by the liver

(D) Is inactivated by spontaneous, organ-independent Hofmann elimination (plus ester hydrolysis), so its clearance does not rely on hepatic or renal function

**Q49.** Terbutaline relaxes bronchial smooth muscle and is also used to delay premature labour. These actions are explained by its property as a:

(A) Non-selective $\alpha$-agonist

(B) Selective $\beta_2$-adrenergic agonist that relaxes bronchial and uterine smooth muscle

(C) Muscarinic antagonist

(D) $\beta_1$-selective agonist used as a cardiac inotrope

**Q50.** Alprazolam, like other benzodiazepines, enhances inhibitory neurotransmission, and its overdose can be reversed by a specific antagonist. The mechanism and antidote are respectively:

(A) Direct opening of the chloride channel; reversed by naloxone

(B) Increased duration of chloride channel opening; reversed by atropine

(C) Positive allosteric modulation of the GABA-A receptor increasing the frequency of chloride channel opening; reversed by flumazenil

(D) Blockade of glutamate NMDA receptors; reversed by physostigmine

**Q51.** Olanzapine, an atypical antipsychotic, causes fewer extrapyramidal effects than haloperidol largely because it:

(A) Has no affinity for dopamine $D_2$ receptors at all

(B) Is a selective $D_2$ agonist

(C) Blocks only muscarinic receptors

(D) Combines moderate $D_2$ blockade with strong 5-$HT_{2A}$ antagonism, which relieves striatal dopamine inhibition (at the cost of metabolic/weight side effects)

**Q52.** Duloxetine relieves both depression and diabetic neuropathic pain because it is a:

(A) Serotonin-noradrenaline reuptake inhibitor (SNRI) that raises synaptic 5-HT and noradrenaline

(B) Selective irreversible monoamine oxidase inhibitor

(C) Pure dopamine $D_2$ antagonist

(D) Selective $\mu$-opioid receptor agonist

**Q53.** Fentanyl is widely used for intra-operative analgesia and in transdermal patches for chronic pain. Compared with morphine, fentanyl is best described as:

(A) A $\kappa$-selective antagonist with no analgesic action

(B) A highly lipophilic $\mu$-opioid agonist roughly 100 times more potent than morphine, with rapid onset and short duration after a bolus

(C) A partial agonist weaker than codeine

(D) An opioid that does not cause respiratory depression

**Q54.** Topiramate is a broad-spectrum antiepileptic (also used in migraine prophylaxis). Among its several actions, an important one that distinguishes it is:

(A) Selective T-type calcium channel block only

(B) Irreversible inhibition of GABA-transaminase

(C) A multimodal action including sodium-channel block, enhancement of GABA-A activity, AMPA/kainate antagonism and carbonic-anhydrase inhibition

(D) Pure NMDA-receptor agonism

**Q55.** Bupivacaine provides long-lasting regional and epidural anaesthesia but is more cardiotoxic than lidocaine because it:

(A) Is highly protein-bound and lipophilic, blocking cardiac sodium channels with slow dissociation, predisposing to refractory ventricular arrhythmias

(B) Opens cardiac potassium channels and slows the heart

(C) Has no effect on nerve sodium channels

(D) Is rapidly metabolised, giving an extremely short duration

**Q56.** Ropinirole is used in early Parkinson's disease and in restless-legs syndrome because it:

(A) Inhibits peripheral DOPA-decarboxylase

(B) Is converted to dopamine within the brain

(C) Irreversibly inhibits monoamine oxidase-B

(D) Is a non-ergot dopamine $D_2/D_3$ receptor agonist that directly stimulates striatal dopamine receptors

**Q57.** Captopril, the first orally active ACE inhibitor, lowers blood pressure by:

(A) Blocking angiotensin II $AT_1$ receptors directly

(B) Inhibiting angiotensin-converting enzyme, lowering angiotensin II and aldosterone while reducing bradykinin breakdown (its sulfhydryl group is linked to taste disturbance and rash)

(C) Blocking L-type calcium channels

(D) Antagonising $\beta_1$-adrenoceptors

**Q58.** Nicorandil relieves angina through a dual mechanism that distinguishes it from a pure nitrate, namely it:

(A) Blocks $\beta_1$-receptors and L-type calcium channels

(B) Inhibits the funny ($I_f$) current in the SA node

(C) Acts both as a nitric-oxide donor (raising cGMP) and as an ATP-sensitive potassium-channel opener, dilating veins and resistance arteries

(D) Inhibits the renin-angiotensin system

**Q59.** Sotalol is a racemic antiarrhythmic whose dominant electrophysiological action places it mainly in Vaughan-Williams Class III. This means it chiefly:

(A) Blocks fast sodium channels to slow conduction (Class I)

(B) Blocks L-type calcium channels in the AV node (Class IV)

(C) Acts only as a pure $\beta$-blocker with no effect on repolarisation

(D) Blocks potassium channels to prolong repolarisation and the action-potential duration (and also has $\beta$-blocking, Class II, activity)

**Q60.** Ticagrelor prevents arterial thrombosis by a mechanism that, unlike clopidogrel, does not require metabolic activation. Ticagrelor:

(A) Reversibly inhibits the platelet $P2Y_{12}$ ADP receptor without needing conversion to an active metabolite

(B) Irreversibly acetylates cyclooxygenase like aspirin

(C) Blocks glycoprotein IIb/IIIa receptors

(D) Activates antithrombin like heparin

**Q61.** Diltiazem, a benzothiazepine calcium-channel blocker, differs from amlodipine in that, in addition to vasodilation, it:

(A) Has no effect on cardiac conduction

(B) Also slows sinoatrial and atrioventricular nodal conduction and reduces heart rate, so it controls supraventricular arrhythmias

(C) Acts only on the venous side to reduce preload

(D) Is an ACE inhibitor

**Q62.** Loratadine is preferred over chlorpheniramine for chronic allergic rhinitis because loratadine:

(A) Blocks $H_2$ receptors and reduces gastric acid

(B) Is a sedating first-generation antihistamine

(C) Is a second-generation $H_1$ antagonist that penetrates the CNS poorly, giving effective allergy relief with little sedation

(D) Acts as a mast-cell stabiliser without receptor blockade

**Q63.** Ketorolac is used as a short-term parenteral analgesic for moderate-to-severe post-operative pain. Its analgesic and anti-inflammatory effects, and its main limitation, arise because it:

(A) Is a selective COX-2 inhibitor with no gastrointestinal risk

(B) Acts only centrally with no peripheral COX inhibition

(C) Is an opioid agonist

(D) Is a potent non-selective COX inhibitor giving strong analgesia, but its inhibition of COX-1 limits use to a few days because of gastrointestinal and renal risk

**Q64.** Probenecid is used for chronic urate lowering in gout (in suitable patients) because it:

(A) Increases renal uric acid excretion by inhibiting its reabsorption at the proximal-tubule urate transporter (a uricosuric)

(B) Inhibits xanthine oxidase and reduces urate production

(C) Inhibits neutrophil microtubule assembly

(D) Is a selective COX-2 inhibitor

**Q65.** Clarithromycin, a macrolide antibiotic, inhibits bacterial growth by:

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

(B) Binding the 50S ribosomal subunit and blocking translocation, inhibiting protein synthesis

(C) Inhibiting DNA gyrase

(D) Inhibiting cell-wall transpeptidases

**Q66.** Rifampicin is a key first-line antitubercular agent that also turns body secretions orange-red. Its bactericidal action is due to:

(A) Inhibition of mycolic-acid synthesis

(B) Inhibition of the 30S ribosomal subunit

(C) Inhibition of bacterial DNA-dependent RNA polymerase, blocking transcription

(D) Inhibition of dihydrofolate reductase

**Q67.** Acyclovir is selectively active against herpes simplex and varicella-zoster viruses because it:

(A) Inhibits the influenza neuraminidase

(B) Blocks viral entry by binding the host CD4 receptor

(C) Inhibits HIV reverse transcriptase after random phosphorylation in all cells

(D) Is first phosphorylated by the viral thymidine kinase, then by host kinases, to a triphosphate that inhibits viral DNA polymerase and terminates the DNA chain

**Q68.** Pioglitazone improves glycaemic control in type 2 diabetes mainly because it:

(A) Activates the nuclear receptor PPAR-$\gamma$, increasing transcription of genes that enhance peripheral insulin sensitivity

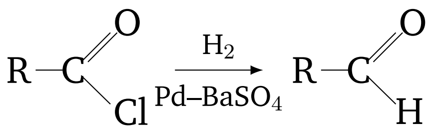
(B) Stimulates pancreatic insulin secretion by closing $K_{ATP}$ channels

(C) Inhibits intestinal $\alpha$-glucosidase

(D) Blocks renal SGLT2 glucose reabsorption

## Part C: Pharmaceutical & Medicinal Chemistry

**Q69.** The catalytic hydrogenation of an acyl chloride over palladium poisoned with barium sulfate (Pd–$BaSO_4$), which stops cleanly at the aldehyde stage, is summarised below.



This selective conversion of an acid chloride to an aldehyde is the:

(A) Stephen reduction

(B) Rosenmund reduction

(C) Clemmensen reduction

(D) Bouveault–Blanc reduction

**Q70.** Treatment of an electron-rich aromatic compound (e.g. *N,N*-dimethylaniline) with a mixture of $POCl_3$ and *N,N*-dimethylformamide (DMF), followed by aqueous work-up, introduces a –CHO group on the ring. This formylation is known as the:

(A) Vilsmeier–Haack reaction

(B) Gattermann–Koch reaction

(C) Kolbe–Schmitt reaction

(D) Houben–Hoesch reaction

**Q71.** A carboxylic acid reacts with hydrazoic acid ($HN_3$) in the presence of a strong acid ($H_2SO_4$) to give a primary amine with one fewer carbon, releasing $N_2$ and $CO_2$ through an acyl azide / nitrene intermediate. This carbon-to-nitrogen migration is the:

(A) Favorskii rearrangement

(B) Pinacol–pinacolone rearrangement

(C) Chichibabin amination

(D) Schmidt reaction

**Q72.** The acid-catalysed conversion shown below proceeds through loss of water to give a carbocation, followed by a 1,2-methyl shift and loss of a proton to form a ketone.

$$(CH_3)_2C(OH)\text{–}C(OH)(CH_3)_2 \xrightarrow[-H_2O]{H^+} (CH_3)_3C\text{–}CO\text{–}CH_3$$

The rearrangement of pinacol to pinacolone illustrated here is the:

(A) Wagner ring expansion

(B) Pinacol–pinacolone rearrangement

(C) Benzilic acid rearrangement

(D) Tiffeneau–Demjanov rearrangement

**Q73.** The conversion of a secondary alcohol into an ester (or other nucleophile-derived product) using diethyl azodicarboxylate (DEAD) and triphenylphosphine, proceeding with clean **inversion** of configuration at the carbinol carbon, is the:

(A) Mitsunobu reaction

(B) Appel reaction

(C) Corey–House synthesis

(D) Tsuji–Trost reaction

**Q74.** Heating pyridine with sodium amide ($NaNH_2$) introduces an amino group specifically at the 2-position of the ring, with evolution of hydrogen gas. This direct amination of an electron-poor azine is the:

(A) Bucherer reaction

(B) Skraup synthesis

(C) Chichibabin reaction

(D) Combes synthesis

**Q75.** Consider the three nitrogen-containing species: (i) pyrrole, (ii) pyridine, (iii) piperidine. Arrange them in order of **increasing** basicity of the nitrogen.

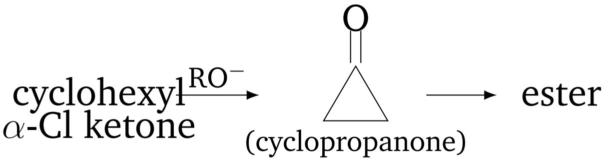
(A) (iii) $<$ (ii) $<$ (i)

(B) (ii) $<$ (iii) $<$ (i)

(C) (i) $<$ (ii) $<$ (iii)

(D) (i) $<$ (iii) $<$ (ii)

**Q76.** An $\alpha$-halo ketone treated with an alkoxide base undergoes ring contraction through a cyclopropanone intermediate to give a ring-contracted carboxylic-acid ester, as outlined below.



This base-induced ring contraction of $\alpha$-halo ketones is the:

(A) Neber rearrangement

(B) Favorskii rearrangement

(C) Meinwald rearrangement

(D) Ramberg–Bäcklund reaction

**Q77.** A nitrile ($R–C\equiv N$) is reduced by anhydrous stannous chloride and HCl to an aldimine salt, which on hydrolysis gives the corresponding aldehyde. This partial reduction of a nitrile to an aldehyde is the:

(A) Stephen reaction

(B) Etard reaction

(C) Gattermann reaction

(D) Sonn–Müller method

**Q78.** In a protic solvent such as methanol, arrange the following nucleophiles in order of **decreasing** nucleophilicity toward a typical $S_N2$ substrate: (i) $F^-$, (ii) $Cl^-$, (iii) $Br^-$, (iv) $I^-$.

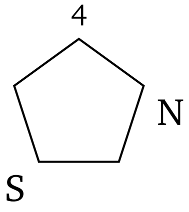
(A) (i) $>$ (ii) $>$ (iii) $>$ (iv)

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

(C) (ii) > (i) > (iv) > (iii)

(D) (iii) > (i) > (ii) > (iv)

**Q79.** The five-membered aromatic heterocycle drawn below contains both a sulfur atom and a nitrogen atom at the 1- and 3-positions; it forms the core of vitamin $B_1$ (thiamine) and of penicillin’s fused bicyclic system.



Identify this heterocycle.

(A) oxazole

(B) isoxazole

(C) imidazole

(D) thiazole

**Q80.** Oxymercuration–demercuration of 3,3-dimethylbut-1-ene gives 3,3-dimethylbutar 2-ol **without** any carbon-skeleton rearrangement. The key reason this differs from acid-catalysed hydration is that oxymercuration:

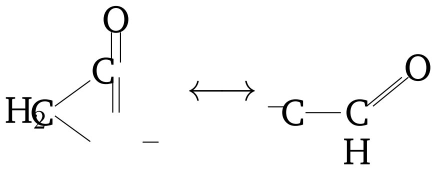
(A) gives the anti-Markovnikov alcohol

(B) proceeds through a free open primary carbocation

(C) proceeds through a bridged mercurinium ion, so no carbocation rearrangement occurs

(D) adds water across the double bond with syn stereochemistry only

**Q81.** The enolate ion derived from acetaldehyde is stabilised by delocalisation of the negative charge over carbon and oxygen, as shown by the two resonance contributors below.



Which statement about the real enolate ion is correct?

(A) The negative charge is fixed entirely on carbon.

(B) It is non-aromatic and therefore not resonance stabilised.

(C) The two contributors are equal in energy and contribute equally.

(D) The charge is delocalised over O and C, with the oxygen-bearing form contributing more (oxygen is more electronegative).

**Q82.** Before catalytic hydrogenation became common, esters were reduced to primary alcohols using sodium metal dissolved in absolute ethanol (a dissolving-metal, single-electron-transfer reduction). This classical reduction of an ester to an alcohol is the:

(A) Bouveault–Blanc reduction

(B) Meerwein–Ponndorf–Verley reduction

(C) Rosenmund reduction

(D) Luche reduction

**Q83.** $\alpha$-D-glucopyranose and $\beta$-D-glucopyranose differ only in the configuration at the hemiacetal (anomeric) carbon, C-1. The correct term for this specific relationship between the two cyclic forms is that they are:

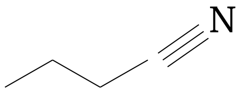
(A) anomers

(B) enantiomers

(C) conformers

(D) structural (constitutional) isomers

**Q84.** Identify the functional group present in the skeletal structure drawn below.



(A) amide

(B) nitrile (cyano group)

(C) amine

(D) imine

**Q85.** Propranolol, atenolol and metoprolol share an aryloxypropanolamine pharmacophore and competitively block $\beta$-adrenergic receptors. To which therapeutic/chemical class do these "-olol" drugs belong?

(A) calcium-channel blockers

(B) angiotensin-converting-enzyme inhibitors

(C) $H_2$-receptor antagonists

(D) $\beta$-adrenergic receptor antagonists ($\beta$-blockers)

**Q86.** Isoniazid (isonicotinic acid hydrazide) is a first-line antitubercular agent. It is a **prodrug** activated by the mycobacterial enzyme KatG, after which it inhibits the synthesis of which cell-wall component of *Mycobacterium tuberculosis*?

(A) mycolic acids

(B) peptidoglycan cross-links

(C) the 30S ribosomal subunit

(D) dihydrofolate

**Q87.** Cyclophosphamide and mechlorethamine exert their cytotoxic action by forming a reactive aziridinium ion that covalently cross-links DNA, chiefly at the N-7 of guanine. These drugs belong to which mechanistic class of anticancer agents?

(A) antimetabolites

(B) topoisomerase inhibitors

(C) bifunctional alkylating agents (nitrogen mustards)

(D) tubulin-binding agents

**Q88.** A drug that binds a receptor and produces a **sub-maximal** response even at full receptor occupancy, and that can antagonise the effect of a full agonist present at the same receptor, is best described as a(n):

(A) full agonist

(B) partial agonist

(C) competitive (neutral) antagonist

(D) inverse agonist

**Q89.** The strongest of the reversible (non-covalent) forces that typically anchors a protonated amine drug to an ionised carboxylate or phosphate residue in its receptor binding site is the:

(A) ionic (electrostatic) interaction

(B) van der Waals (London dispersion) force

(C) hydrophobic interaction

(D) dipole–dipole interaction

**Q90.** Esomeprazole (the *S*-enantiomer of omeprazole) and levocetirizine (the active *R*-enantiomer of cetirizine) were marketed after the corresponding racemate. The development strategy of relaunching a single, more favourable enantiomer of an already-marketed racemic drug is called:

(A) bioisosteric replacement

(B) scaffold hopping

(C) prodrug design

(D) a chiral (racemic) switch

**Q91.** Warfarin, a 4-hydroxycoumarin oral anticoagulant, produces its effect by inhibiting which enzyme, thereby blocking the $\gamma$-carboxylation of clotting factors II, VII, IX and X?

(A) cyclooxygenase (COX)

(B) vitamin K epoxide reductase (VKORC1)

(C) thrombin (factor IIa) directly

(D) HMG-CoA reductase

**Q92.** Vancomycin, a glycopeptide antibiotic active against Gram-positive bacteria, acts at a different site from the $\beta$-lactams. It binds with high affinity to which structural motif, thereby blocking transglycosylation/transpeptidation in cell-wall synthesis?

(A) the 50S ribosomal subunit

(B) DNA gyrase

(C) the terminal D-Ala–D-Ala of the peptidoglycan precursor

(D) dihydropteroate synthase

**Q93.** A drug suspension contains drug in excess of its solubility, so the concentration of dissolved drug in solution stays essentially constant as it degrades. The degradation of the dissolved drug therefore follows kinetics that are:

(A) first order with respect to total drug

(B) second order

(C) apparent zero order

(D) fractional (1.5) order

**Q94.** According to the Arrhenius equation $k = A\,e^{-E_a/RT}$, if the rate constant of a reaction **doubles** when the temperature is raised, this behaviour is explained primarily by:

(A) the exponential increase in the fraction of molecules possessing energy $\geq E_a$

(B) a large increase in the average kinetic energy of all molecules

(C) a decrease in the activation energy $E_a$ with temperature

(D) an increase in the enthalpy change $\Delta H$ of the reaction

**Q95.** Which pharmaceutical inorganic compound is used both as a systemic antacid and to alkalinise the urine (e.g. to enhance excretion of acidic drugs in overdose), being absorbed and producing systemic alkalosis if overused?

(A) calcium carbonate

(B) sodium bicarbonate

(C) aluminium hydroxide

(D) magnesium trisilicate

**Q96.** The industrial oxidation of ethylene (ethene) to acetaldehyde using a palladium(II) chloride / copper(II) chloride catalyst system in water, with $O_2$ as the terminal oxidant, is the:

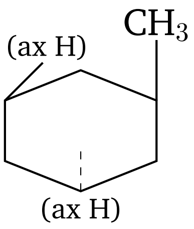
(A) Heck reaction

(B) Oppenauer oxidation

(C) Wacker process (Wacker–Tsuji oxidation)

(D) Etard reaction

**Q97.** In methylcyclohexane the equatorial conformer is favoured over the axial conformer (by about 7.5 $\text{kJ mol}^{-1}$). The chair below shows the **axial** methyl, which suffers destabilising interactions with the two axial hydrogens on the same face.



These strain-producing interactions between the axial substituent and the axial hydrogens at C-3 and C-5 are called:

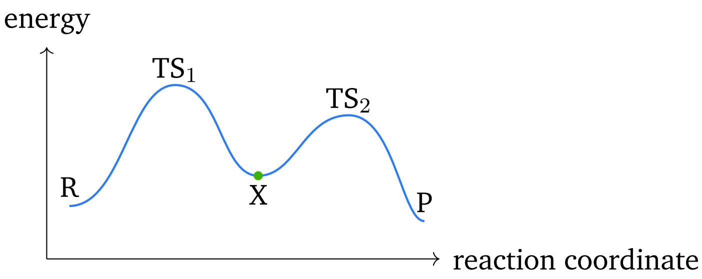
(A) torsional (eclipsing) strain

(B) angle (Baeyer) strain

(C) gauche butane strain only

(D) 1,3-diaxial interactions

**Q98.** The reaction-coordinate diagram below shows a two-step reaction. Point **X** sits in the valley between the two energy maxima.



Which statement about point **X** and the rate-determining step is correct?

(A) X is a transition state and the second step is rate-determining.

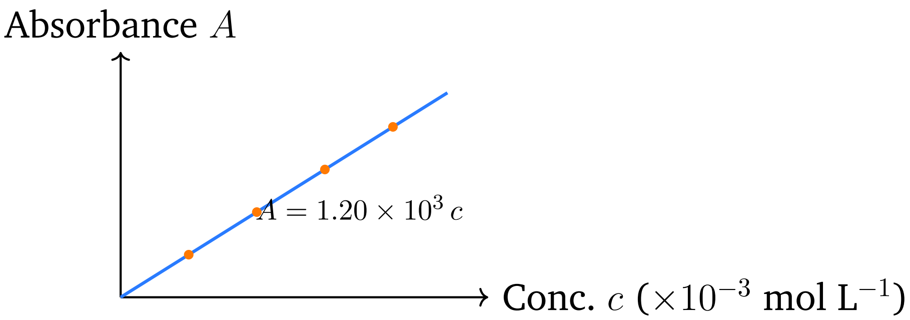
(B) X is a transition state and the first step is rate-determining.

(C) X is a reaction intermediate and the second step (TS$_2$) is rate-determining.

(D) X is a reaction intermediate, and the first step (with the higher barrier TS$_1$) is rate-determining.

## Part D: Pharmaceutical Analysis & Quality Assurance

**Q99.** The Beer–Lambert calibration line below was recorded at $\lambda_{max}$ in a 1.0 cm cell. The best-fit line is $A = 1.20 \times 10^3\,c$, with $c$ in mol L$^{-1}$ and the line passing through the origin. Taking the path length as $l = 1.0$ cm, the molar absorptivity $\varepsilon$ of the drug is:



(A) $1.20 \times 10^3$ mol $L^{-1}$ $cm^{-1}$

(B) $0.83 \times 10^{-3}$ L $mol^{-1}$ $cm^{-1}$

(C) $1.20 \times 10^3$ L $mol^{-1}$ $cm^{-1}$

(D) $1.20$ L $mol^{-1}$ $cm^{-1}$

**Q100.** In bromatometry the half-reaction of the titrant is $BrO_3^- + 6H^+ + 6e^- \rightarrow Br^- + 3H_2O$. For a $0.0250$ M potassium bromate ($KBrO_3$) solution used under these strongly acidic conditions, the normality is:

(A) 0.150 N

(B) 0.0250 N

(C) 0.0500 N

(D) 0.00417 N

**Q101.** In a modern Karl Fischer moisture titration, the endpoint is most commonly detected electrically by the technique known as:

(A) Visual fading of a phenolphthalein colour

(B) Dead-stop (biamperometric) endpoint detection

(C) Gravimetric drying to constant mass

(D) Refractive-index matching

**Q102.** To assay a metal ion that forms only a weak complex with EDTA and gives no sharp visual endpoint, the analyte is treated with the magnesium–EDTA complex so that it liberates an equivalent amount of $Mg^{2+}$, which is then titrated. This strategy is a:

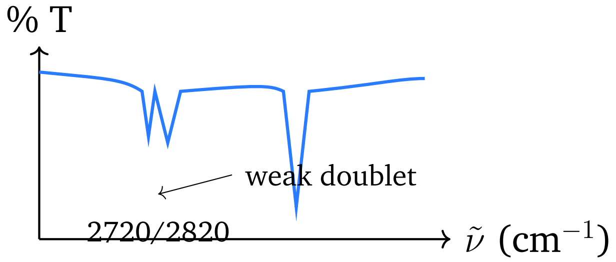
(A) Direct titration

(B) Back (residual) titration

(C) Masking titration

(D) Displacement (substitution) titration

**Q103.** The schematic IR spectrum below shows, in addition to a strong carbonyl band, a pair of weak bands near **2720 and 2820 cm**$^{-1}$ (the marked feature). This characteristic doublet, just below the normal C–H region, is the fingerprint of which functional group?



(A) A tertiary amine

(B) An ether linkage

(C) An aldehyde (–CHO)

(D) A sulphonic acid

**Q104.** In the Andrews titration, potassium iodate in concentrated hydrochloric acid oxidises the analyte, and at the endpoint iodine is converted to iodine monochloride. The iodate half-reaction at the endpoint is best represented as:

(A) $IO_3^- \rightarrow I^-$ (6-electron change)

(B) $IO_3^- \rightarrow I_2$ (5-electron change)

(C) $IO_3^- \rightarrow IO_4^-$ (oxidation of iodate)

(D) $IO_3^- \rightarrow ICl$ (4-electron change, iodine in +1 state)

**Q105.** On a TLC plate the solvent front advanced 12.0 cm from the origin while a steroid spot moved 4.8 cm from the origin. The retardation factor $R_f$ of the steroid is:

(A) 0.40

(B) 2.50

(C) 0.48

(D) 0.60

**Q106.** A peak elutes with retention time $t_R = 6.0$ min and a baseline (tangent) width $w = 0.30$ min. Using $N = 16\,(t_R/w)^2$, the number of theoretical plates is approximately:

(A) 320

(B) 1600

(C) 6400

(D) 400

**Q107.** In fluorescence spectrometry the measured fluorescence intensity $F$ is directly proportional to the analyte concentration $c$ only when:

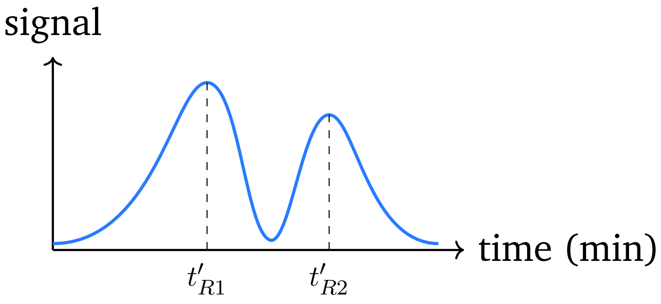
(A) The solution is highly concentrated and strongly absorbing

(B) The solution is dilute, so that absorbance is low and inner-filter effects are negligible

(C) The excitation wavelength equals the emission wavelength

(D) The quantum yield is exactly zero

**Q108.** For the two peaks in the chromatogram below the adjusted retention times are $t'_{R1} = 2.0$ min and $t'_{R2} = 3.0$ min (measured from the void-volume marker). The selectivity (separation) factor $\alpha = t'_{R2}/t'_{R1}$ between the two solutes is:



(A) 0.67

(B) 1.0

(C) 5.0

(D) 1.5

**Q109.** In a $^1$H NMR spectrum, the protons of a simple benzene ring (aromatic C–H) typically resonate in which chemical-shift region?

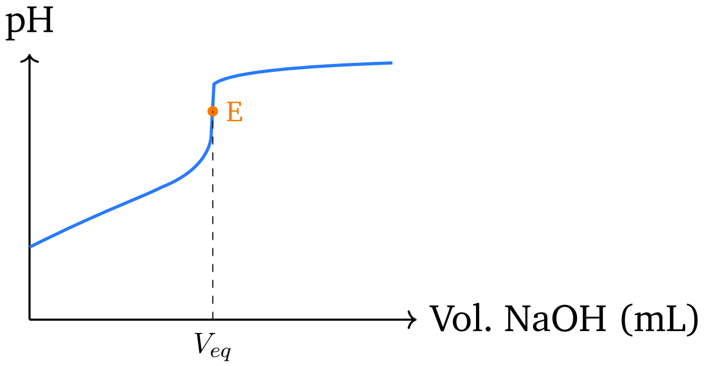
(A) About $\delta$ 7–8 ppm (deshielded by the aromatic ring current)

(B) About $\delta$ 0–0.5 ppm (more shielded than TMS)

(C) About $\delta$ 2–2.5 ppm (same as a methyl ketone)

(D) About $\delta$ 12–13 ppm (same as a carboxylic acid OH)

**Q110.** The curve below is for the titration of a *weak* acid with a strong base (NaOH). At the equivalence point marked **E**, the pH of the solution is:



(A) Exactly 7.0, the same as a strong acid–strong base titration

(B) Below 7 (acidic), because excess acid remains

(C) Above 7 (basic), because the conjugate base of the weak acid hydrolyses

(D) Independent of the nature of the acid

**Q111.** An organic compound containing a single bromine atom shows, in its mass spectrum, molecular-ion peaks at M and M+2 with an intensity ratio of very nearly **1:1**. This pattern arises because the two stable isotopes of bromine are:

(A) $^{79}$Br only, so there is no M+2 peak

(B) $^{79}$Br and $^{81}$Br, present in almost equal natural abundance

(C) $^{35}$Br and $^{37}$Br in a 3:1 ratio

(D) Three isotopes giving an M, M+1, M+2 triplet

**Q112.** In flame atomic absorption spectroscopy (AAS), the principal function of the burner/nebuliser (atomiser) assembly is to:

(A) Disperse the radiation into its component wavelengths

(B) Generate the element-specific resonance line

(C) Detect and amplify the transmitted radiation

(D) Convert the sample into a population of free, ground-state gaseous atoms

**Q113.** In gas chromatography, the Kovats retention index expresses the retention behaviour of a solute relative to a homologous series of:

(A) $n$-alkanes, with each $n$-alkane assigned an index of 100 × its carbon number

(B) Primary alcohols, indexed by boiling point

(C) Aromatic hydrocarbons, indexed alphabetically

(D) Fatty-acid methyl esters, indexed by molecular weight

**Q114.** EDTA reacts with metal ions in a strict 1:1 molar ratio. If 25.0 mL of $0.0100$ M EDTA is required to titrate a calcium solution to the endpoint, the number of millimoles of Ca$^{2+}$ present in the sample is:

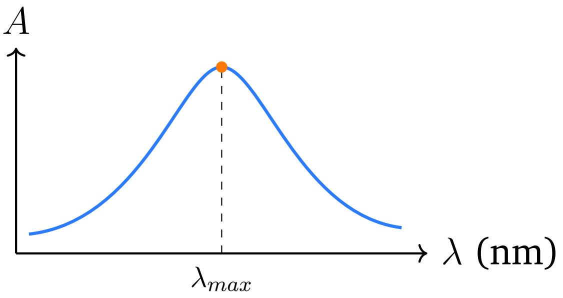
(A) 0.500 mmol

(B) 2.50 mmol

(C) 0.250 mmol

(D) 0.0250 mmol

**Q115.** The UV curve below shows a single absorption band with its maximum marked $\lambda_{max}$. The unsaturated group (such as C=O or an aromatic ring) that is directly responsible for this electronic absorption band is correctly termed a:



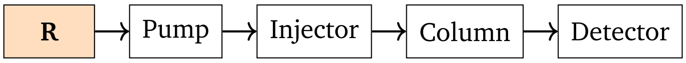
(A) An auxochrome

(B) A chromophore

(C) A bathochrome

(D) A fluorophore quencher

**Q116.** In the liquid-chromatograph block diagram below, identify the component labelled **R**, which is positioned at the very start of the flow path and holds the mobile-phase solvent before it is drawn into the pump:



(A) The detector cell

(B) The analytical column

(C) The sample-injection valve

(D) The mobile-phase (solvent) reservoir

**Q117.** Phenolphthalein is a suitable visual indicator for the titration of a weak acid with a strong base because its colour-change (transition) interval lies in the pH range:

(A) About 8.0–10.0, changing colourless to pink

(B) About 3.1–4.4, changing red to yellow

(C) About 0–2, changing blue to green

(D) About 12–14, changing pink to colourless

**Q118.** When a sodium salt is aspirated into a flame in flame photometry, the intense yellow emission used for its quantitation arises from the sodium D lines at a wavelength of approximately:

(A) 422 nm (a violet line)

(B) 589 nm (the yellow D doublet)

(C) 766 nm (a red line)

(D) 254 nm (an ultraviolet line)

**Q119.** In size-exclusion chromatography, a solute's elution volume $V_e$ is related to the void volume $V_0$ (totally excluded, largest species) and the total permeation volume $V_t$ (smallest species). A molecule that can partially enter the pores will elute with a volume that:

(A) Is less than $V_0$

(B) Equals $V_0$ exactly, regardless of size

(C) Lies between $V_0$ and $V_t$, increasing as the molecule gets smaller

(D) Is greater than $V_t$

**Q120.** Tetramethylsilane (TMS) is chosen as the internal reference ($\delta = 0$) in $^1$H NMR spectroscopy chiefly because it:

(A) Absorbs strongly in the ultraviolet at 254 nm

(B) Contains an unpaired electron giving a sharp signal

(C) Resonates far downfield of nearly all organic protons

(D) Is inert, volatile, and its 12 equivalent protons give a single sharp peak upfield of almost all other organic protons

**Q121.** In a coulometric assay carried out at 100% current efficiency, the amount of analyte reacted is calculated directly from the total charge passed, using:

(A) Faraday's laws of electrolysis ($Q = nFN$, charge $\propto$ moles reacted)

(B) Beer's law ($A = \varepsilon cl$)

(C) The Henderson–Hasselbalch equation

(D) The van Deemter equation

**Q122.** Under ICH stability-testing guidelines, the standard *accelerated* storage condition used to support a shelf-life claim for a typical solid oral product is:

(A) $5\,^\circ$C with no humidity control

(B) $40\,^\circ$C $\pm\,2\,^\circ$C and 75% $\pm\,5$% relative humidity for 6 months

(C) $-20\,^\circ$C for 12 months

(D) $60\,^\circ$C and 0% relative humidity for 1 month

## Part E: Pharmacognosy & Natural Products

**Q123.** A pharmacopoeial index lists crude drugs strictly in the order *Acacia*, *Belladonna*, *Cinchona*, *Digitalis*, *Ergota*, ignoring botany, chemistry and use. This arrangement of crude drugs follows which basis of classification?

(A) Morphological classification

(B) Alphabetical classification

(C) Chemical classification

(D) Serological classification

**Q124.** Grouping crude drugs strictly by their botanical position, for example placing all Solanaceae members (*Belladonna*, *Stramonium*, *Hyoscyamus*) together because they share the same family, is termed:

(A) Chemical classification

(B) Pharmacological classification

(C) Taxonomical (botanical) classification

(D) Morphological classification

**Q125.** Under the morphological scheme, crude drugs are sorted by the plant part used. Which of the following is an **organized** leaf drug rather than an unorganized exudate?

(A) Tragacanth (a dried gum)

(B) Benzoin (a balsamic resin)

(C) Catechu (a dried aqueous extract)

(D) Buchu (the dried leaf of *Barosma betulina*)

**Q126.** Yohimbine, an indole alkaloid with peripheral $\alpha_2$-adrenergic blocking and aphrodisiac use, is obtained chiefly from the bark of which botanical source?

(A) *Pausinystalia yohimbe*

(B) *Erythroxylum coca*

(C) *Papaver somniferum*

(D) *Claviceps purpurea*

**Q127.** Ergotamine, used as an oxytocic and in migraine, is one of the peptide ergoline alkaloids produced by a fungal sclerotium that parasitises the ovary of rye. This drug is:

(A) Opium (latex of *Papaver somniferum*)

(B) Vinca (*Catharanthus roseus* herb)

(C) Ergot (sclerotium of *Claviceps purpurea* on *Secale cereale*)

(D) Ipecacuanha (root of *Cephaelis ipecacuanha*)

**Q128.** Cocaine, the local-anaesthetic tropane alkaloid from the leaves of *Erythroxylum coca*, is a di-ester of ecgonine. On alkaline hydrolysis cocaine yields ecgonine together with methanol and which aromatic acid (recognised by its characteristic odour)?

(A) Salicylic acid

(B) Benzoic acid

(C) Cinnamic acid

(D) Gallic acid

**Q129.** Among the opium alkaloids, papaverine is pharmacologically distinct from morphine because it is a non-narcotic smooth-muscle relaxant (antispasmodic/vasodilator). Chemically papaverine belongs to which alkaloidal class?

(A) Benzylisoquinoline alkaloid

(B) Phenanthrene (morphinan) alkaloid

(C) Tropane alkaloid

(D) Purine alkaloid

**Q130.** The dried flowering herb of *Convallaria majalis* (lily of the valley) contains a cardioactive glycoside whose aglycone strophanthidin bears a five-membered butenolide ring and whose sugar is L-rhamnose. This cardiac glycoside is:

(A) Sennoside A

(B) Glycyrrhizin

(C) Sinigrin

(D) Convallatoxin

**Q131.** Aloin (barbaloin), the chief purgative principle of aloes, resists ordinary acid hydrolysis because its anthranol sugar is attached directly through a carbon–carbon bond rather than through oxygen. Aloin is therefore classified as:

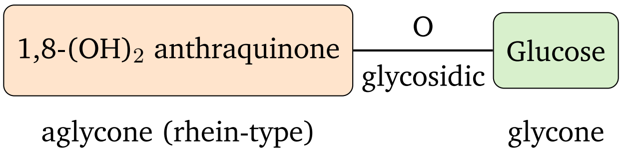
(A) An O-glycoside hydrolysable by dilute acid

(B) A C-glycoside (anthrone C-glucoside)

(C) An N-glycoside

(D) An S-glycoside

**Q132.** The schematic shows a 1,8-dihydroxyanthraquinone aglycone joined through oxygen at C-8 to a glucose unit, the type of glycoside that gives rhubarb and senna their purgative action.



The class of natural compounds represented by this aglycone is:

(A) Cardenolide steroids

(B) Cyanogenetic glycosides

(C) Anthraquinone (hydroxyanthracene) derivatives

(D) Saponin triterpenes

**Q133.** Baicalin, a yellow constituent of the root of *Scutellaria baicalensis* (Chinese skullcap), is a flavone joined to glucuronic acid. The non-sugar (aglycone) portion baicalein therefore belongs to which class of polyphenolic compounds?

(A) Flavonoids (a flavone aglycone)

(B) Alkaloids

(C) Cardiac glycosides

(D) Bitter terpenoid principles

**Q134.** The volatile oil distilled from *Thymus vulgaris* owes its antiseptic action chiefly to a phenolic monoterpenoid that is a positional isomer of carvacrol. This chief constituent is:

(A) Thymol

(B) Eugenol

(C) Geraniol

(D) Citral

**Q135.** Rosemary oil, distilled from the leaves of *Rosmarinus officinalis*, is rich in a bicyclic monoterpene ether (an oxide) that is also the chief constituent of eucalyptus oil. This component is:

(A) Anethole

(B) 1,8-Cineole (eucalyptol)

(C) Menthol

(D) Safrole

**Q136.** Copaiba is a pathological oleoresin that exudes from the trunk of South American *Copaifera* species. Being an oleoresin, it consists essentially of resin dissolved in which natural co-solvent fraction?

(A) Gum and mucilage

(B) A fixed (fatty) oil

(C) Benzoic and cinnamic acids

(D) Its own volatile (essential) oil

**Q137.** Guar gum, obtained from the endosperm of *Cyamopsis tetragonoloba* seeds and used as a binder and bulk laxative, is chemically a galactomannan. On hydrolysis it therefore yields a backbone and side-chain built from which two monosaccharides?

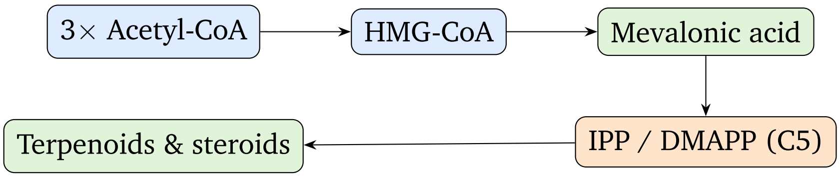
(A) D-Mannose and D-galactose

(B) D-Glucose and L-rhamnose

(C) D-Galacturonic acid and L-arabinose

(D) D-Glucuronic acid and D-xylose

**Q138.** The biosynthetic scheme below builds the universal C5 isoprene unit used for plant terpenoids and steroids. Identify the pathway depicted.



(A) Shikimic acid pathway

(B) Acetate–mevalonate (mevalonic acid) pathway

(C) Acetate–malonate pathway

(D) Pentose phosphate pathway

**Q139.** The shikimic acid pathway condenses erythrose-4-phosphate with phosphoenolpyruvate and proceeds via shikimate and chorismate. Which group of secondary metabolites is the **principal** product of this aromatic pathway?

(A) Acyclic monoterpenes

(B) Saturated fatty acids

(C) Phenylpropanoids and aromatic amino acids

(D) Reducing sugars

**Q140.** In the standardisation of a crude drug a weighed sample is charred, the carbonised mass is moistened with dilute *sulphuric acid*, and the whole is ignited to constant weight. The ash value reported by this sulphuric-acid procedure is the:

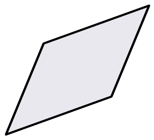
(A) Total ash value

(B) Acid-insoluble ash

(C) Water-soluble ash

(D) Sulphated ash

**Q141.** The microscopical sketch below shows a single, well-defined monoclinic calcium-oxalate crystal of the kind found scattered in the parenchyma of many barks (for example cinnamon and cinchona). This isolated crystal form is named a:



calcium oxalate

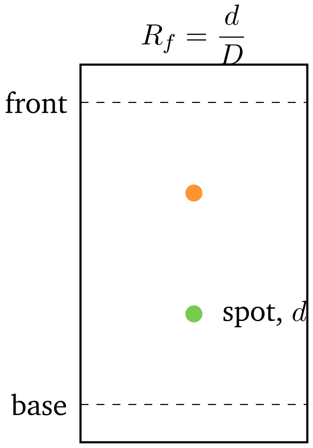
(A) Rosette (cluster) crystal

(B) Raphide bundle

(C) Prism crystal

(D) Crystal sand (microsphenoidal mass)

**Q142.** The TLC plate below profiles a herbal extract, showing the baseline, two resolved spots, and the solvent front.



If the lower spot migrates $d = 2.4$ cm while the solvent front $D$ advances $4.0$ cm from the baseline, the $R_f$ value of that spot is:

(A) 0.60

(B) 1.67

(C) 0.24

(D) 0.40

## Part F: Pharmaceutical Biotechnology & Microbiology

**Q143.** Type II restriction endonucleases may leave 5′ overhangs, 3′ overhangs, or blunt ends. Which one of the following enzymes cleaves its palindrome so as to leave **3′ recessed / 3′ single-stranded overhangs**?

(A) *Pst*I, which cuts CTGCA↓G leaving 3′ overhangs

(B) *Bam*HI, which cuts G↓GATCC leaving 5′ overhangs

(C) *Sma*I, which cuts CCC↓GGG leaving blunt ends

(D) *Hpa*I, which cuts GTT↓AAC leaving blunt ends

**Q144.** A genomic library is to be built from inserts of roughly 150 kb. A standard plasmid (a few kb) and a cosmid ($\sim$45 kb) cannot stably carry such a large fragment. The vector of choice for cloning these very large inserts is the:

(A) Phagemid (M13-derived)

(B) Standard high-copy plasmid such as pUC19

(C) Bacterial artificial chromosome (BAC), based on the F-factor replicon

(D) $\lambda$ insertion vector

**Q145.** To amplify a eukaryotic transcript that lacks introns, the messenger RNA is first copied into complementary DNA, which is then amplified. The enzyme that synthesises the first cDNA strand from the mRNA template in this RT-PCR step is:

(A) *Taq* DNA polymerase

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

(C) Terminal deoxynucleotidyl transferase

(D) RNA polymerase II

**Q146.** Which one of the following recombinant therapeutic proteins is used as a disease-modifying agent in **relapsing–remitting multiple sclerosis**?

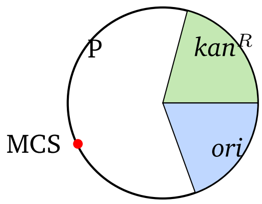
(A) Interferon-$\beta$ (interferon beta-1a / beta-1b)

(B) Recombinant human insulin

(C) Recombinant factor VIII

(D) Recombinant erythropoietin

**Q147.** The circular expression vector shown carries an origin of replication, a kanamycin-resistance gene ($kan^R$), a strong promoter (P), and a multiple cloning site (MCS). After transformation into *E. coli*, the role of the $kan^R$ gene during selection is to:



(A) Provide the origin from which replication starts

(B) Drive transcription of the cloned insert

(C) Act as the site where foreign DNA is inserted

(D) Allow only transformed cells to grow on kanamycin-containing medium

**Q148.** Industrial production of **lactic acid** by submerged fermentation of glucose or whey, used to make biodegradable polylactic acid and food acidulants, is most commonly carried out using:

(A) *Aspergillus niger*

(B) *Lactobacillus delbrueckii* (homofermentative lactic acid bacteria)

(C) *Saccharomyces cerevisiae*

(D) *Acetobacter aceti*

**Q149.** Vitamin $B_2$ (**riboflavin**) for pharmaceutical and feed use is produced on an industrial scale by overproducing fermentation with the ascomycete fungus:

(A) *Penicillium chrysogenum*

(B) *Streptomyces griseus*

(C) *Ashbya gossypii*

(D) *Clostridium acetobutylicum*

**Q150.** **Dextran**, a branched glucose polymer used as a plasma volume expander, is produced from sucrose by the extracellular enzyme dextransucrase of:

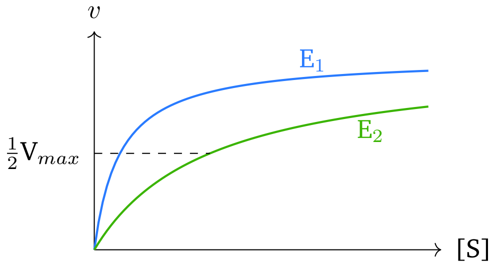
(A) *Leuconostoc mesenteroides*

(B) *Escherichia coli*

(C) *Bacillus subtilis*

(D) *Candida utilis*

**Q151.** Two enzymes, $E_1$ and $E_2$, act on the same substrate; their Michaelis–Menten curves are shown. $E_1$ reaches half of its $V_{max}$ at a much lower [S] than $E_2$. Therefore, with respect to this substrate:



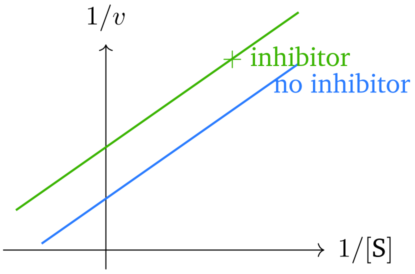
(A) $E_2$ has the higher affinity because its $K_m$ is larger

(B) $E_1$ has the higher affinity because its $K_m$ is smaller

(C) Both enzymes have identical affinity since $V_{max}$ is the same

(D) Affinity cannot be compared from a Michaelis–Menten plot

**Q152.** The double-reciprocal (Lineweaver–Burk) plot below shows an uninhibited reaction and one with an **uncompetitive inhibitor** added. The two lines are *parallel*. This pattern indicates that the inhibitor:



(A) Raises $V_{max}$ while leaving $K_m$ unchanged

(B) Increases apparent $K_m$ but leaves $V_{max}$ unchanged (competitive pattern)

(C) Lowers both apparent $K_m$ and $V_{max}$ by the same factor, giving parallel lines

(D) Leaves $K_m$ unchanged but lowers $V_{max}$ (non-competitive pattern)

**Q153.** Which immunoglobulin class is the principal antibody of **mucosal secretions** (saliva, tears, colostrum, intestinal fluid), where it exists as a dimer joined by a J chain and protected by a secretory component?

(A) IgG

(B) IgM

(C) IgE

(D) Secretory IgA

**Q154.** In the international nomenclature of therapeutic monoclonal antibodies, the sub-stem in the name tells the source of the antibody. A **fully human** monoclonal antibody (for example adalimumab) is identified by the sub-stem:

(A) `-omab` (fully murine)

(B) `-ximab` (chimeric)

(C) `-umab` (fully human)

(D) `-zumab` (humanised)

**Q155.** Of the three complement-activation routes, the **alternative pathway** differs from the classical pathway in that it:

(A) Is triggered by antigen–antibody (IgG/IgM) immune complexes binding C1q

(B) Is initiated directly on microbial surfaces by spontaneous C3 hydrolysis, without antibody, and is stabilised by factors B, D and properdin

(C) Begins with mannose-binding lectin recognising sugars on the pathogen

(D) Does not lead to formation of the membrane-attack complex

**Q156.** Serum sickness and the Arthus reaction, in which soluble antigen–antibody (immune) complexes deposit in vessel walls and joints, activate complement, and recruit neutrophils causing tissue damage, are examples of which Gell and Coombs hypersensitivity type?

(A) Type I (immediate, IgE-mediated)

(B) Type II (antibody-mediated cytotoxic)

(C) Type IV (delayed, T-cell-mediated)

(D) Type III (immune-complex-mediated)

**Q157.** The bacterial **capsule** is demonstrated microscopically by a negative-staining method (for example India ink or the Anthony method) in which the capsule appears as:

(A) A clear, unstained halo around the cell against a dark background

(B) A deeply stained terminal spore within the cell

(C) A network of stained flagella radiating from the cell

(D) Stained granules of poly-$\beta$-hydroxybutyrate inside the cell

**Q158.** In thermal sterilisation kinetics, the **z-value** of a microbial population is defined as the:

(A) Time at a fixed temperature to reduce the viable count by 90% (one log cycle)

(B) Temperature change (in °C) required to change the decimal-reduction time D by a factor of ten

(C) Number of organisms present before sterilisation

(D) Total heat delivered, expressed as equivalent minutes at 121°C

**Q159.** Bacterial endotoxins (pyrogens, the lipopolysaccharide of Gram-negative cell walls) are heat-stable and survive ordinary autoclaving. Glass vials and ampoules are therefore **depyrogenated** by:

(A) Autoclaving at 121°C for 15 min

(B) Filtration through a 0.22 $\mu$m membrane

(C) Dry heat at 250°C for 30 min (or an equivalent validated cycle)

(D) Exposure to ethylene oxide gas

**Q160.** The Limulus amoebocyte lysate (LAL) test, used to detect bacterial endotoxin in parenteral products and water for injection, works because the lysate from the horseshoe-crab amoebocyte:

(A) Lyses any viable bacteria present in the sample

(B) Changes colour in proportion to the total protein content

(C) Detects fungal $\beta$-glucans only, not endotoxin

(D) Forms a gel (clots) on contact with endotoxin through a clotting-enzyme cascade

## Detailed Solutions

### Q1.

**Solution**

**Concept — Solubility product of a 1:2 salt:** For $Ag_2CrO_4 \rightleftharpoons 2\,Ag^+ + CrO_4^{2-}$, if molar solubility $= s$ then $[Ag^+] = 2s$ and $[CrO_4^{2-}] = s$. **Reasoning:** $K_{sp} = [Ag^+]^2[CrO_4^{2-}] = (2s)^2(s) = 4s^3$. **Why the other options are wrong:**

- (A) $s^2$ is the expression for a 1:1 salt (e.g. AgCl).
- (B) $s^3$ ignores the factor of 2 from the two $Ag^+$ ions.
- (D) $27s^4$ applies to a 1:3 salt such as $AB_3$.

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

**Answer: (C)** Go Back to Q1

### Q2.

**Solution**

**Concept — log P:** The logarithm (base 10) of the partition coefficient measures lipophilicity; $\log P = \log_{10} P$. **Reasoning:** $P = 1000 = 10^3$, so $\log P = \log_{10}(10^3) = 3$. **Why the other options are wrong:**

- (B) 2 corresponds to $P = 100$.
- (C) 10 confuses $P$ with the exponent base.
- (D) 1 corresponds to $P = 10$.

**Final Answer:** $\log P = \log 10^3 = 3 \Rightarrow \boxed{A}$

**Answer: (A)** Go Back to Q2

### Q3.

**Solution**

**Concept — HLB of a blend:** The blend HLB is the weight-fraction (algebraic) average of the component HLB values: $\text{HLB}_{mix} = \sum f_i\,\text{HLB}_i$. **Reasoning:** $\text{HLB} = 0.40(15) + 0.60(4.3) = 6.0 + 2.58 = 8.58 \approx 8.6$. **Why the other options are wrong:**

- (A) 9.7 over-weights the high-HLB surfactant.
- (C) 10.3 inverts the weight fractions.
- (D) 4.3 is just the Span 80 value, ignoring the blend.

**Final Answer:** $0.4(15) + 0.6(4.3) = 8.6 \Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q3

## Q4.

### Solution

**Concept — Newtonian liquid:** A straight $\tau$ vs $\dot{\gamma}$ line through the origin means viscosity $\eta = \tau/\dot{\gamma}$ is the same at every shear rate. **Reasoning:** A constant viscosity independent of shear rate, with no yield value, defines a Newtonian liquid (e.g. water, glycerin, light mineral oil). **Why the other options are wrong:**

- (B) Pseudoplastic liquids show falling viscosity with shear (curve bows to the stress axis).
- (C) Dilatant liquids show rising viscosity with shear.
- (D) Thixotropy is a time-dependent (hysteresis) effect, not a single straight line.

**Final Answer:** constant $\eta$, line through origin $\Rightarrow$ Newtonian $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q4

## Q5.

### Solution

**Concept — Dilatant flow:** In shear-thickening systems the apparent viscosity *increases* with shear rate, so the flow curve bows toward the shear-rate axis. **Reasoning:** Concentrated deflocculated suspensions of small particles (e.g. starch pastes, 50%+ solids) dilate and resist flow more strongly as shear rises; this is dilatant (shear-thickening) flow. **Why the other options are wrong:**

- (A) Newtonian flow is a straight line through the origin.
- (B) Pseudoplastic is the opposite (shear-thinning) behaviour.
- (D) Plastic flow has a yield value, so the curve starts off the origin on the stress axis.

**Final Answer:** viscosity rises with shear $\Rightarrow$ dilatant $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q5

## Q6.

### Solution

**Concept — Kinematic viscosity:** $\nu = \eta/\rho$ (dynamic viscosity divided by density). Its CGS unit is the stoke. **Reasoning:** Dimensionally $\eta/\rho$ has units (g cm$^{-1}$ s$^{-1}$)/(g cm$^{-3}$) $=$ cm$^2$/s. Hence 1 stoke $=$ 1 cm$^2$/s. **Why the other options are wrong:**

- (A) Poise is the unit of *dynamic* (absolute) viscosity, not kinematic.
- (C) Pa·s is the SI unit of dynamic viscosity.
- (D) dyne/cm is a unit of surface tension.

**Final Answer:** $1\ \text{stoke} = 1\ \text{cm}^2/\text{s} \Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q6

## Q7.

### Solution

**Concept — Carr's index:** Carr's % $= \dfrac{\rho_{tapped} - \rho_{bulk}}{\rho_{tapped}} \times 100$. **Reasoning:** $= \dfrac{0.625 - 0.50}{0.625} \times 100 = \dfrac{0.125}{0.625} \times 100 = 20\%$. **Why the other options are wrong:**

- (B) 25% wrongly divides the difference by the bulk density.
- (C) 12.5% multiplies the raw difference (0.125) by 100 only.
- (D) 16% is a miscalculation of the ratio.

**Final Answer:** $(0.125/0.625) \times 100 = 20\% \Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q7

## Q8.

### Solution

**Concept — Angle of repose:** The angle of repose correlates with powder flow: $< 30^\circ$ indicates excellent/good flow, 31–35° good, 36–40° fair, 41–45° passable, $> 56^\circ$ very poor. **Reasoning:** 28° lies below 30°, so the powder shows excellent (good) flow. **Why the other options are wrong:**

- (A) Very poor flow needs angles above about 56°.
- (B) Passable flow is around 41–45°.
- (C) Poor flow is around 46–55°.

**Final Answer:** $28^\circ < 30^\circ \Rightarrow$ excellent flow $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q8

## Q9.

**Solution**

**Concept — Mode of a frequency distribution:** The modal class is the size interval with the tallest bar (highest frequency). **Reasoning:** The tallest bar (height 2.9) is centred on the 18 label, so the most frequent class is the 18–28 $\mu$m interval. **Why the other options are wrong:**

- (A) 8–18 is the smaller adjacent class with a much shorter bar.
- (C) 28–38 has a lower frequency than the modal class.
- (D) 48–58 is in the tail with the lowest frequency.

**Final Answer:** tallest bar $\Rightarrow$ 18–28 $\mu$m $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q9

## Q10.

**Solution**

**Concept — Henderson–Hasselbalch:** $\text{pH} = \text{pK}_a + \log\dfrac{[\text{salt}]}{[\text{acid}]}$. **Reasoning:** salt:acid $= 1:10$, so $\log(1/10) = -1$; $\text{pH} = 6.10 + (-1) = 5.10$. **Why the other options are wrong:**

- (A) 6.10 ignores the unequal ratio (assumes 1:1).
- (B) 7.10 wrongly adds 1 (treats ratio as 10:1).
- (D) 4.10 subtracts 2 (uses ratio 1:100).

**Final Answer:** $\text{pH} = 6.10 - 1 = 5.10 \Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q10

## Q11.

**Solution**

**Concept — Spreading coefficient:** $S = \gamma_S - (\gamma_L + \gamma_{SL})$; spontaneous spreading needs the work of adhesion to exceed the work of cohesion. **Reasoning:** When $S \geq 0$ (positive) the liquid wets and spreads over the solid/liquid surface; a higher solid surface tension favours spreading. **Why the other options are wrong:**

- (B) A negative $S$ means the liquid beads up and does not spread.
- (C) $\gamma_{SL} = 0$ alone does not guarantee a positive $S$.
- (D) A maximal liquid surface tension raises cohesion, opposing spreading.

**Final Answer:** spreading occurs when $S \geq 0$ (positive) $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q11

## Q12.

**Solution**

**Concept — Stability constant:** A larger formation constant $K$ means the equilibrium lies further toward the complex, i.e. a more stable (more strongly associated) complex. **Reasoning:** $K_X = 10^4 > K_Y = 10^2$, so complex X is 100 times more stable than Y. **Why the other options are wrong:**

- (A) Y has the smaller $K$, so it is less stable.
- (B) Different $K$ values mean unequal stability.
- (C) A smaller $K$ means a weaker, not stronger, complex.

**Final Answer:** higher $K$ (X) $\Rightarrow$ more stable complex $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** 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 $t$ is linear with slope $-k$. **Reasoning:** The figure shows ln C decreasing linearly with time, the signature of first-order decay (e.g. many hydrolysing drugs in solution). **Why the other options are wrong:**

- (A) Zero order gives a straight line for $C$ (not ln C) versus $t$.
- (C) Second order gives a straight line for $1/C$ versus $t$.

- (D) Third order linearises $1/C^2$ versus $t$.

**Final Answer:** linear ln C vs t ⇒ first order ⇒ $\boxed{\text{B}}$

**Answer: (B)** Go Back to Q13

## Q14.

**Solution**

**Concept — Zero-order shelf life:** For zero order, $t_{90} = \dfrac{0.1\,C_0}{k_0}$ (time to lose 10% of the initial concentration). **Reasoning:** $t_{90} = \dfrac{0.1 \times 40}{0.2} = \dfrac{4}{0.2} = 20$ days. **Why the other options are wrong:**

- (A) 4 days forgets to divide by $k_0$.
- (B) 40 days uses the full $C_0$ instead of 10%.
- (D) 200 days misplaces a decimal in $k_0$.

**Final Answer:** $t_{90} = (0.1 \times 40)/0.2 = 20$ days ⇒ $\boxed{\text{C}}$

**Answer: (C)** Go Back to Q14

## Q15.

**Solution**

**Concept — $Q_{10}$ temperature coefficient:** A rise of $n \times 10$°C multiplies the rate by $Q_{10}^n$. **Reasoning:** For a 20°C rise, $n = 2$, so the rate increases by $Q_{10}^2 = 3^2 = 9$-fold.

**Why the other options are wrong:**

- (A) 3-fold is the effect of only a single 10°C step.
- (B) 6-fold wrongly multiplies $Q_{10}$ by 2 instead of squaring.
- (C) 30-fold has no basis in the $Q_{10}$ relationship.

**Final Answer:** $3^2 = 9$-fold ⇒ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q15

## Q16.

### Solution

**Concept — Langmuir isotherm:** Rearranging $\frac{x}{m} = \frac{abC}{1+bC}$ gives $\frac{C}{(x/m)} = \frac{1}{ab} + \frac{C}{a}$, which is linear in $C$. **Reasoning:** Therefore a plot of $C/(x/m)$ versus $C$ is a straight line; its slope $(1/a)$ and intercept $(1/ab)$ give the Langmuir constants. **Why the other options are wrong:**

- (B) $\log(x/m)$ vs $\log C$ linearises the *Freundlich* isotherm, not Langmuir.
- (C) $x/m$ vs $C$ is the curved (non-linear) plot.
- (D) $x/m$ vs $1/C$ does not linearise the Langmuir form.

**Final Answer:** plot $C/(x/m)$ vs $C \Rightarrow \boxed{\text{A}}$

**Answer: (A)** **Go Back to Q16**

## Q17.

### Solution

**Concept — Eutectic point:** The point where two liquidus curves meet at the lowest temperature, at which the whole mixture melts/freezes at one constant temperature, is the eutectic point. **Reasoning:** At E the solid of fixed composition melts completely without leaving residue; eutectic mixtures (e.g. lidocaine–prilocaine) are exploited in topical anaesthetic creams. **Why the other options are wrong:**

- (A) A peritectic point involves a solid reacting with liquid to form a new solid, not a simple minimum.
- (B) The inversion temperature relates to emulsion phase inversion, not phase diagrams.
- (D) A critical solution point appears in partially miscible liquid systems.

**Final Answer:** lowest meeting point E $\Rightarrow$ eutectic point $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** **Go Back to Q17**

## Q18.

### Solution

**Concept — Noyes–Whitney, surface area:** Dissolution rate is directly proportional to the effective surface area $A$ exposed to the medium. **Reasoning:** Micronisation reduces particle size and so multiplies the total surface area $A$, increasing $dC/dt$. This is why poorly soluble (BCS II) drugs are often micronised. **Why the other options are wrong:**

- (A) $C_s$ (saturation solubility) is essentially unchanged by particle size (except for very fine sub-micron particles).
- (C) Increasing $h$ would *slow* dissolution, and micronisation does not increase $h$.
- (D) The diffusion coefficient $D$ is a property of the drug/medium, not particle size.

**Final Answer:** micronisation raises $A \Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q18

## Q19.

### Solution

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

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

**Final Answer:** $Q \propto \sqrt{t} \Rightarrow$ Higuchi model $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q19

## Q20.

### Solution

**Concept — Korsmeyer–Peppas exponent (sphere):** For a spherical matrix, $n \leq 0.43$ indicates Fickian (diffusion-controlled) release; $0.43 < n < 0.85$ is anomalous; $n = 0.85$ is Case-II. **Reasoning:** With $n \leq 0.43$ the drug leaves the sphere mainly by diffusion through the matrix, i.e. Fickian diffusion. **Why the other options are wrong:**

- (B) Case-II (zero-order, polymer relaxation) corresponds to $n = 0.85$ for a sphere.
- (C) Super Case-II requires $n > 0.85$.
- (D) Anomalous (non-Fickian) transport is the intermediate range $0.43 < n < 0.85$.

**Final Answer:** $n \leq 0.43$ (sphere) $\Rightarrow$ Fickian diffusion $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q20

## Q21.

### Solution

**Concept — USP dissolution apparatus:** Apparatus 1 is the rotating basket; Apparatus 2 is the paddle; Apparatus 3 is the reciprocating cylinder; Apparatus 4 is the flow-through cell. **Reasoning:** The basket (Apparatus 1) holds the dosage form and is preferred for capsules or floating formulations that would otherwise drift on the paddle. **Why the other options are wrong:**

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

**Final Answer:** USP Apparatus 1 $\Rightarrow$ rotating basket $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q21

## Q22.

**Solution**

**Concept — First-pass metabolism:** Drug absorbed from the gut passes through the liver before reaching systemic circulation; metabolism there lowers the fraction reaching the blood intact. **Reasoning:** Extensive presystemic (first-pass) metabolism reduces the oral bioavailability $F$, often making oral doses much larger than intravenous ones. **Why the other options are wrong:**

- (A) Absolute bioavailability can never exceed 1 (100%).
- (C) High first-pass implies high, not zero, hepatic clearance.
- (D) First-pass metabolism reduces the systemic fraction; it does not raise net absorption.

**Final Answer:** extensive first-pass $\Rightarrow$ oral $F$ reduced $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q22

## Q23.

**Solution**

**Concept — Tablet binder:** Binders impart cohesiveness so that granules and the final tablet hold together. Starch paste (mucilage) is a classic wet-granulation binder. **Reasoning:** Starch paste coats the powder particles and, on drying, glues them into robust granules, giving adequate tablet mechanical strength. **Why the other options are wrong:**

- (B) Lubricants (e.g. magnesium stearate) reduce die-wall friction.
- (C) Glidants (e.g. colloidal silica) improve powder flow.
- (D) Film-coating polymers (e.g. HPMC) form the outer coat, not the granule bond.

**Final Answer:** starch paste $\Rightarrow$ binder $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q23

## Q24.

### Solution

**Concept — Weight variation:** The acceptable range is the average weight $\pm$ the percentage limit. **Reasoning:** $\pm 5\%$ of 500 mg = $\pm 25$ mg, giving a range of $500 - 25 = 475$ mg to $500 + 25 = 525$ mg. **Why the other options are wrong:**

- (A) 490–510 corresponds to a $\pm 2\%$ limit.
- (C) 450–550 corresponds to a $\pm 10\%$ limit.
- (D) 495–505 corresponds to a $\pm 1\%$ limit.

**Final Answer:** $500 \pm 25$ mg $\Rightarrow$ 475–525 mg $\Rightarrow$ $\boxed{\text{B}}$

$\boxed{\textbf{Answer: (B)}}$ Go Back to Q24

## Q25.

### Solution

**Concept — Capsule sizing:** For hard-gelatin capsules the larger the size number, the smaller the capacity; sizes run 000 (largest) down to 5 (smallest) for human use. **Reasoning:** Size 5 holds the least powder ($\approx 0.13$ mL) and is the smallest commonly used human size. **Why the other options are wrong:**

- (A) 000 is the *largest* human capsule.
- (B) 0 is a large/medium size.
- (D) 1 is larger than 5.

**Final Answer:** smallest human size $\Rightarrow$ size 5 $\Rightarrow$ $\boxed{\text{C}}$

$\boxed{\textbf{Answer: (C)}}$ Go Back to Q25

## Q26.

### Solution

**Concept — Displacement value (DV):** Base needed = (mould capacity $\times$ number) $-$ (total drug $\div$ DV). **Reasoning:** Total drug $= 12 \times 0.3 = 3.6$ g; base displaced $= 3.6/1.5 = 2.4$ g; nominal base $= 12 \times 2 = 24$ g; base required $= 24 - 2.4 = 21.6$ g. **Why the other options are wrong:**

- (A) 24.0 g ignores the displacement correction entirely.
- (B) 20.4 g subtracts the full 3.6 g instead of $3.6/1.5$.
- (C) 23.6 g subtracts only $3.6/1.5$ from the wrong nominal base.

**Final Answer:** $24 - (3.6/1.5) = 24 - 2.4 = 21.6\text{ g} \Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q26

## Q27.

### Solution

**Concept — Ointment base classes:** Absorption bases are anhydrous (or contain little water) but can soak up aqueous fluids while staying greasy; lanolin is the classic example. **Reasoning:** Anhydrous wool fat takes up roughly twice its weight of water to form a w/o emulsion yet remains oily, the defining property of an absorption base. **Why the other options are wrong:**

- (B) Oleaginous bases (e.g. white petrolatum) take up almost no water.
- (C) Water-soluble (PEG) bases contain no oily phase and wash off completely.
- (D) Water-removable o/w bases are creams with water as the external phase.

**Final Answer:** lanolin $\Rightarrow$ absorption base $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q27

## Q28.

### Solution

**Concept — Sedimentation volume:** $F = \dfrac{V_u}{V_o}$ (ultimate sediment volume / original suspension volume). **Reasoning:** $F = 12/30 = 0.40$. Values nearer 1 indicate a more flocculated, pharmaceutically acceptable suspension. **Why the other options are wrong:**

- (A) 0.60 inverts the sediment and clear-supernatant volumes.
- (C) 2.50 inverts the ratio ($V_o/V_u$).
- (D) 0.12 misreads the original volume.

**Final Answer:** $F = 12/30 = 0.40 \Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q28

## Q29.

### Solution

**Concept — Creaming:** Creaming is the reversible upward (or downward) migration of dispersed droplets to form a concentrated layer, redispersible by shaking, with no permanent phase separation. **Reasoning:** Oil droplets (less dense) rise and concentrate at the top but remain individual droplets, so gentle agitation restores a uniform emulsion. This is the definition of creaming. **Why the other options are wrong:**

- (A) Cracking (breaking) is irreversible coalescence with permanent phase separation.
- (B) Phase inversion converts o/w to w/o (or vice versa).
- (D) Ostwald ripening is the growth of large droplets at the expense of small ones, a distinct mechanism.

**Final Answer:** reversible droplet layering ⇒ creaming ⇒ $\boxed{\text{C}}$

$\boxed{\textbf{Answer: (C)}}$ Go Back to Q29

## Q30.

### Solution

**Concept — Moist-heat sterilisation:** The pharmacopoeial reference autoclave cycle uses saturated steam under pressure. **Reasoning:** The standard cycle is 121°C for 15 minutes at 15 psi (about 103 kPa) gauge pressure, which reliably kills spores including *Geobacillus stearothermophilus*. **Why the other options are wrong:**

- (A) 160°C for 2 h is a *dry-heat* (hot-air oven) cycle.
- (C) 180°C for 30 min is also a dry-heat condition.
- (D) 100°C for 30 min (boiling) does not reliably destroy bacterial spores.

**Final Answer:** 121°C, 15 min, 15 psi ⇒ $\boxed{\text{B}}$

$\boxed{\textbf{Answer: (B)}}$ Go Back to Q30

## Q31.

### Solution

**Concept — Ophthalmic tonicity:** Eye drops should match the tonicity of tears, equivalent to a 0.9% w/v sodium chloride (normal saline) solution. **Reasoning:**

Lacrimal fluid has an osmolality close to 0.9% NaCl; the comfortable tolerable range is roughly 0.6–2.0% NaCl equivalent, but 0.9% is the ideal isotonic value. **Why the other options are wrong:**

- (B) 5.0% would be strongly hypertonic and irritating.
- (C) 0.09% is grossly hypotonic.
- (D) 2.5% is hypertonic and lies at the edge of tolerability.

**Final Answer:** isotonic with tears ⇒ 0.9% w/v NaCl ⇒ $\boxed{\text{A}}$

**Answer: (A)** Go Back to Q31

## Q32.

### Solution

**Concept — Tablet hardness:** Crushing strength is the diametral force needed to fracture a tablet, measured with a hardness tester (Monsanto, Pfizer, Strong-Cobb or modern motorised types). **Reasoning:** The Monsanto/Pfizer-type tester

applies a measured load across the tablet diameter and reads the force at fracture in kg-force or newtons. **Why the other options are wrong:**

- (A) The Roche friabilator measures friability (abrasion loss), not crushing strength.
- (C) The disintegration apparatus times tablet break-up in fluid.
- (D) The Andreasen pipette is used for particle-size analysis by sedimentation.

**Final Answer:** crushing strength ⇒ Monsanto/Pfizer hardness tester ⇒ $\boxed{\text{B}}$

**Answer: (B)** Go Back to Q32

## Q33.

### Solution

**Concept — Reynolds number:** For Newtonian flow in a circular pipe, $Re < 2100$ is laminar, $Re > 4000$ is fully turbulent, and the range in between is transitional. **Reasoning:** The unstable transition where flow shifts from smooth streamlines to chaotic eddies lies at roughly $Re = 2100\text{–}4000$. **Why the other options are wrong:**

- (A) $Re < 100$ is well within the laminar regime.
- (B) $Re = 1.0$ is deeply laminar (creeping flow).
- (D) $Re > 100000$ is strongly turbulent, far past the transition.

**Final Answer:** transitional $Re \approx 2100\text{–}4000 \Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q33

## Q34.

### Solution

**Concept — Niosomes:** Niosomes are bilayer vesicles formed by hydration of non-ionic surfactants (e.g. Spans, Tweens) usually together with cholesterol, entrapping both hydrophilic and lipophilic drugs. **Reasoning:** Unlike liposomes (phospholipid-based), niosomes use non-ionic surfactants, giving greater chemical stability and lower cost while serving a similar carrier role. **Why the other options are wrong:**

- (A) Phospholipid bilayers describe liposomes, not niosomes.
- (B) Solid lipid nanoparticles have a solid (crystalline) lipid core, not surfactant bilayers.
- (C) Albumin microspheres are protein matrix particles, a different carrier class.

**Final Answer:** non-ionic surfactant bilayer vesicles $\Rightarrow$ niosomes $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q34

## Q35.

### Solution

**Concept — Volume of distribution:** $V_d$ is the proportionality constant relating the amount of drug in the body to the plasma concentration, $V_d = \text{Dose}/C_0$. **Reasoning:** For an IV bolus in a one-compartment model, $C_0$ is the concentration extrapolated to $t = 0$. Here $V_d = 750\,\text{mg}/30\,\text{mg/L} = 25$ L. **Why the other options are wrong:**

- (A) 0.04 L inverts the ratio ($C_0/\text{Dose}$).

- (B) 22.5 L is not obtainable from the data.
- (D) 250 L misplaces the decimal point.

**Final Answer:** $V_d = 750/30 = 25$ L $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** **Go Back to Q35**

## Q36.

### Solution

**Concept — Half-life from rate constant:** For first-order elimination the slope of the $\ln C$ vs $t$ line is $-k_e$, and $t_{1/2} = 0.693/k_e$. **Reasoning:** $k_e = 0.0578\,\text{h}^{-1}$, so $t_{1/2} = 0.693/0.0578 \approx 12$ h. **Why the other options are wrong:**

- (A) 6 h corresponds to $k_e \approx 0.116\,\text{h}^{-1}$.
- (C) 24 h corresponds to $k_e \approx 0.029\,\text{h}^{-1}$.
- (D) 0.69 h wrongly takes $t_{1/2} = 0.693$ regardless of $k_e$.

**Final Answer:** $t_{1/2} = 0.693/0.0578 \approx 12$ h $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** **Go Back to Q36**

## Q37.

### Solution

**Concept — Absolute bioavailability:** For equal oral and IV doses, $F = \text{AUC}_{oral}/\text{AUC}_{IV}$. **Reasoning:** $F = 18/45 = 0.40$, i.e. 40% of the oral dose reaches the systemic circulation intact. **Why the other options are wrong:**

- (B) 0.18 ignores the IV AUC.
- (C) 0.45 uses the IV AUC value itself.
- (D) 2.5 inverts the ratio ($\text{AUC}_{IV}/\text{AUC}_{oral}$).

**Final Answer:** $F = 18/45 = 0.40 \Rightarrow \boxed{\text{A}}$

**Answer: (A)** **Go Back to Q37**

## Q38.

### Solution

**Concept — Two-compartment model:** A biphasic semi-log decline reflects an early distribution ($\alpha$) phase followed by a slower elimination ($\beta$) phase. **Reasoning:** After an IV bolus the drug first redistributes from the central compartment (blood and well-perfused organs) into peripheral tissues, giving the steep early fall; the shallower terminal segment then reflects true elimination. This two-segment pattern defines a two-compartment model. **Why the other options are wrong:**

- (A) Zero-order gives a curved (not log-linear) plot.
- (B) Flip-flop kinetics applies to slow oral absorption, not an IV bolus.
- (C) Michaelis-Menten saturation produces a concave (non-linear) semi-log curve, not two straight segments.

**Final Answer:** Biphasic = two-compartment distribution then elimination $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q38

## Q39.

### Solution

**Concept — pH partitioning / ion trapping:** Only the un-ionised, lipid-soluble form of a drug crosses membranes readily; the ionised form is trapped where it forms. **Reasoning:** A weak acid (p$K_a$ 3.5) is largely un-ionised in acidic gastric fluid, so it is absorbed there; once in the more alkaline plasma it ionises and is "trapped". Absorption thus depends on the un-ionised fraction set by the Henderson-Hasselbalch relationship. **Why the other options are wrong:**

- (A) Passive pH-dependent diffusion, not active transport, governs this.
- (C) The ionised form penetrates membranes poorly.
- (D) The un-ionised form crosses readily, so the drug is not impermeable.

**Final Answer:** Un-ionised form absorbed, ion-trapped where more ionised $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q39

## Q40.

### Solution

**Concept — Potency vs efficacy:** A leftward-shifted curve with the same plateau indicates greater potency at equal efficacy. **Reasoning:** P and Q reach the same $E_{max}$ (equal efficacy), but P achieves any given effect at a lower dose (its curve is to the left), so P has the lower $\text{ED}_{50}$ and is the more potent drug. **Why the other options are wrong:**

- (A) Efficacy is the plateau, which is equal here.
- (B) Q lies to the right, so it is less potent, not more.
- (D) Both reach the same maximum, so neither is a partial agonist.

**Final Answer:** P more potent, equal efficacy $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q40

## Q41.

### Solution

**Concept — Non-competitive antagonism:** A non-competitive (insurmountable) antagonist lowers the maximal response, which extra agonist cannot restore. **Reasoning:** The progressive fall in the plateau that more agonist cannot overcome is the defining feature of a non-competitive or irreversible antagonist, since the effective number of available receptors is reduced. **Why the other options are wrong:**

- (B) A competitive antagonist shifts the curve right in parallel without lowering $E_{max}$.
- (C) A partial agonist itself produces a response and does not simply depress an agonist curve in this way.
- (D) A chemical antagonist inactivates the agonist in solution, not by this curve pattern.

**Final Answer:** Depressed, unrestorable maximum = non-competitive $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q41

## Q42.

**Solution**

**Concept — Intrinsic activity (efficacy):** Intrinsic activity measures the ability of a bound drug to activate its receptor. **Reasoning:** It is scaled from 0 to 1: a full agonist has intrinsic activity 1, a pure antagonist 0, and a partial agonist a value between. Affinity (binding) and intrinsic activity (activation) are distinct properties. **Why the other options are wrong:**

- (A) That describes affinity, not intrinsic activity.
- (C) The dose for 50% effect is the $\mathrm{ED}_{50}$ (potency).
- (D) The $-\log$ giving a dose ratio of 2 is $\mathrm{pA}_2$ for an antagonist.

**Final Answer:** Ability of bound drug to activate the receptor (0 to 1) $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q42

## Q43.

**Solution**

**Concept — Therapeutic index:** $\mathrm{TI} = \mathrm{LD}_{50}/\mathrm{ED}_{50}$; a larger value means a wider safety margin. **Reasoning:** $\mathrm{TI} = 200/5 = 40$. A high index indicates that the lethal dose is far above the effective dose. **Why the other options are wrong:**

- (A) 5 is the $\mathrm{ED}_{50}$, not the ratio.
- (B) 0.025 inverts the ratio ($\mathrm{ED}_{50}/\mathrm{LD}_{50}$).
- (C) 195 subtracts the doses instead of dividing.

**Final Answer:** $\mathrm{TI} = 200/5 = 40 \Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q43

## Q44.

**Solution**

**Concept — Receptor tyrosine kinases:** Kinase-linked receptors autophosphorylate tyrosine residues that recruit SH2-domain signalling proteins. **Reasoning:** Insulin and epidermal growth factor bind receptor tyrosine kinases; ligand binding causes dimerisation and autophosphorylation, creating docking sites for SH2-domain effectors (e.g. the Ras-MAPK and PI3K pathways). This is the highlighted branch. **Why the other options are wrong:**

- (A) The nicotinic receptor is a ligand-gated ion channel.
- (B) The $\beta_2$-receptor is a GPCR ($G_s$/cAMP).
- (D) Cortisol acts on an intracellular nuclear receptor.

**Final Answer:** Insulin/EGF act on receptor tyrosine kinases $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q44

## Q45.

**Solution**

**Concept — Organophosphate AChE inhibition:** Organophosphates phosphorylate the esteratic site of acetylcholinesterase, forming a very stable complex. **Reasoning:** Unlike the carbamate neostigmine, which inhibits the enzyme reversibly for minutes to hours, echothiophate phosphorylates it to give a long-lasting (effectively irreversible) block. The complex can "age", and early treatment with the oxime reactivator pralidoxime can regenerate the enzyme before ageing. **Why the other options are wrong:**

- (A) A few-minute reversible action describes edrophonium, not an organophosphate.
- (C) It strongly inhibits the enzyme.
- (D) It is not a nicotinic antagonist.

**Final Answer:** Stable phosphorylation that can age; pralidoxime reactivates $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q45

## Q46.

**Solution**

**Concept — Short-acting antimuscarinic mydriatics:** Tropicamide is a rapid, short-acting muscarinic antagonist used to dilate the pupil for examination. **Reasoning:** Blocking iris and ciliary muscarinic receptors gives mydriasis and cycloplegia that resolve within a few hours, whereas atropine's effect can last days. The short duration is the practical advantage for routine fundoscopy. **Why the other options are wrong:**

- (A) It blocks, not stimulates, muscarinic receptors.
- (B) It is reversible and short-acting, not irreversible.
- (D) Antimuscarinics cause mydriasis, not miosis.

**Final Answer:** Short-acting antimuscarinic with brief mydriasis/cycloplegia $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q46

## Q47.

### Solution

**Concept — Esmolol:** Esmolol is an ultra-short-acting $\beta_1$-selective blocker. **Reasoning:** Its ester linkage is rapidly hydrolysed by red-cell esterases, giving a half-life of about 9 minutes. This makes it ideal for titratable, quickly reversible control of heart rate and blood pressure during surgery or hypertensive emergencies. **Why the other options are wrong:**

- (B) It is $\beta_1$-selective and very short-acting, not long-acting and non-selective.
- (C) It is a blocker, not a $\beta_2$-agonist.
- (D) It blocks reversibly, not by alkylation.

**Final Answer:** Ester-hydrolysed $\beta_1$-blocker with ultra-short action $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q47

## Q48.

### Solution

**Concept — Cisatracurium / Hofmann elimination:** Cisatracurium is a non-depolarising blocker cleared by organ-independent degradation. **Reasoning:** At body pH and temperature it breaks down spontaneously by Hofmann elimination (with some ester hydrolysis), so its clearance does not depend on hepatic or renal function. This makes it predictable in organ failure, and it produces less laudanosine than atracurium. **Why the other options are wrong:**

- (A) It is non-depolarising, not a pseudocholinesterase substrate like succinylcholine.
- (B) It is not eliminated unchanged by the kidney.
- (C) It does not rely on biliary/hepatic excretion.

**Final Answer:** Cleared by organ-independent Hofmann elimination $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q48

## Q49.

**Solution**

**Concept — $\beta_2$-agonist terbutaline:** Selective $\beta_2$-agonists relax bronchial and uterine smooth muscle. **Reasoning:** Terbutaline stimulates $\beta_2$-adrenoceptors, raising cAMP in smooth muscle. This relieves bronchospasm in asthma and relaxes the uterus, so it is also used as a tocolytic to delay premature labour. **Why the other options are wrong:**

- (A) It is not an $\alpha$-agonist.
- (C) It is an agonist, not a muscarinic antagonist.
- (D) It is $\beta_2$-selective, not a $\beta_1$ cardiac inotrope.

**Final Answer:** Selective $\beta_2$-agonist relaxing bronchial and uterine muscle $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q49

## Q50.

**Solution**

**Concept — Benzodiazepine mechanism and antidote:** Benzodiazepines are positive allosteric modulators of GABA-A; flumazenil is their specific antagonist. **Reasoning:** Alprazolam binds an allosteric site on the GABA-A receptor-chloride channel and increases the *frequency* of chloride channel opening in response to GABA. Flumazenil, a competitive benzodiazepine-site antagonist, reverses this and is used in overdose. **Why the other options are wrong:**

- (A) They cannot open the channel without GABA; naloxone reverses opioids.
- (B) Increasing channel-open duration is the barbiturate action; atropine is not the antidote.
- (D) They modulate GABA-A, not NMDA receptors.

**Final Answer:** Increases $Cl^-$ channel opening frequency; reversed by flumazenil $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q50

## Q51.

**Solution**

**Concept — Atypical antipsychotic:** Olanzapine combines $D_2$ blockade with strong 5-$HT_{2A}$ antagonism. **Reasoning:** Serotonin (5-$HT_{2A}$) blockade in the nigrostriatal pathway relieves dopamine suppression there, so fewer extrapyramidal effects occur than with haloperidol. The trade-off is prominent metabolic side effects (weight gain, dyslipidaemia). **Why the other options are wrong:**

- (A) It does block $D_2$ receptors, just with added 5-$HT_{2A}$ blockade.
- (B) It is a $D_2$ antagonist, not an agonist.
- (C) Muscarinic blockade is a side-effect profile, not the antipsychotic mechanism.

**Final Answer:** $D_2$ plus strong 5-$HT_{2A}$ blockade $\Rightarrow \boxed{\text{D}}$

Answer: (D) **Go Back to Q51**

## Q52.

**Solution**

**Concept — SNRI duloxetine:** Duloxetine inhibits reuptake of both serotonin and noradrenaline. **Reasoning:** By blocking the SERT and NET transporters it raises synaptic 5-HT and noradrenaline. The noradrenergic component contributes to its efficacy in diabetic and other neuropathic pain as well as depression. **Why the other options are wrong:**

- (B) It is not an MAO inhibitor.
- (C) It is not a dopamine $D_2$ antagonist.
- (D) It is not an opioid agonist.

**Final Answer:** Serotonin-noradrenaline reuptake inhibitor $\Rightarrow \boxed{\text{A}}$

Answer: (A) **Go Back to Q52**

## Q53.

**Solution**

**Concept — Fentanyl:** Fentanyl is a highly lipophilic, very potent $\mu$-opioid agonist. **Reasoning:** It is roughly 100 times more potent than morphine, with rapid onset because of high lipid solubility and short duration after a bolus due to redistribution. These properties suit intra-operative use and transdermal delivery for chronic pain. **Why the other options are wrong:**

- (A) It is a potent agonist, not a $\kappa$ antagonist.
- (C) It is far more potent than codeine, not a weak partial agonist.
- (D) Like all $\mu$-agonists it causes dose-dependent respiratory depression.

**Final Answer:** Lipophilic $\mu$-agonist $\sim 100\times$ morphine potency $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q53

## Q54.

**Solution**

**Concept — Topiramate:** Topiramate is a multimodal antiepileptic. **Reasoning:**

It blocks voltage-gated sodium channels, enhances GABA-A-mediated inhibition, antagonises AMPA/kainate glutamate receptors and inhibits carbonic anhydrase. This broad action underlies its efficacy across seizure types and in migraine prophylaxis. **Why the other options are wrong:**

- (A) T-type calcium block alone describes ethosuximide.
- (B) Irreversible GABA-transaminase inhibition is vigabatrin.
- (D) It does not act as an NMDA agonist.

**Final Answer:** Multimodal ($Na^+$, GABA, AMPA, carbonic anhydrase) $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q54

## Q55.

**Solution**

**Concept — Bupivacaine cardiotoxicity:** Bupivacaine is a long-acting, lipophilic, highly protein-bound local anaesthetic. **Reasoning:** It blocks cardiac sodium

channels and dissociates slowly ("fast-in, slow-out"), so it accumulates in cardiac tissue and can cause refractory ventricular arrhythmias and cardiac arrest if injected intravascularly. Its high lipid solubility also gives its long duration of nerve block. **Why the other options are wrong:**

- (B) It blocks sodium, not opens potassium, channels.
- (C) It does block nerve sodium channels (that is how it anaesthetises).
- (D) Its duration is long, not extremely short.

**Final Answer:** Slow-dissociating cardiac $Na^+$ block causing arrhythmias ⇒ $\boxed{\text{A}}$

**Answer: (A)** Go Back to Q55

## Q56.

### Solution

**Concept — Ropinirole:** Ropinirole is a non-ergot dopamine receptor agonist. **Reasoning:** It directly stimulates striatal $D_2/D_3$ receptors, bypassing the need for dopamine synthesis, which is useful in early Parkinson's disease and in restless-legs syndrome. **Why the other options are wrong:**

- (A) Peripheral DOPA-decarboxylase inhibition describes carbidopa.
- (B) It is not converted to dopamine; it acts on the receptor directly.
- (C) MAO-B inhibition describes selegiline/rasagiline.

**Final Answer:** Non-ergot $D_2/D_3$ agonist ⇒ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q56

## Q57.

### Solution

**Concept — Captopril (ACE inhibitor):** Captopril inhibits angiotensin-converting enzyme. **Reasoning:** Reduced angiotensin II lowers vasoconstriction and aldosterone, while reduced bradykinin breakdown adds vasodilation. Its sulfhydryl (-SH) group is associated with taste disturbance and skin rash, distinguishing it among ACE inhibitors. **Why the other options are wrong:**

- (A) Direct $AT_1$ blockade describes ARBs (e.g. losartan).
- (C) It is not a calcium-channel blocker.
- (D) It is not a $\beta$-blocker.

**Final Answer:** ACE inhibition; sulfhydryl group ⇒ $\boxed{\text{B}}$

**Answer: (B)** Go Back to Q57

## Q58.

**Solution**

**Concept — Nicorandil:** Nicorandil is a nitrate-plus-potassium-channel-opener antianginal. **Reasoning:** It donates nitric oxide (raising cGMP, dilating veins to reduce preload) and opens ATP-sensitive potassium channels (hyperpolarising vascular smooth muscle to dilate resistance arteries, reducing afterload). This dual action distinguishes it from a pure nitrate. **Why the other options are wrong:**

- (A) It is not a $\beta$-blocker or calcium-channel blocker.
- (B) Funny-current inhibition describes ivabradine.
- (D) It does not act on the renin-angiotensin system.

**Final Answer:** NO donor plus $K_{ATP}$-channel opener $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q58

## Q59.

**Solution**

**Concept — Sotalol (Class III):** Sotalol prolongs repolarisation by blocking potassium channels and also blocks $\beta$-receptors. **Reasoning:** Its dominant Class III action prolongs the action-potential duration and effective refractory period via $K^+$ channel block, while the additional $\beta$-blocking (Class II) activity slows the heart. Excess repolarisation prolongation can cause QT prolongation and torsades de pointes. **Why the other options are wrong:**

- (A) Fast $Na^+$ block is Class I.
- (B) L-type calcium block is Class IV.
- (C) It does prolong repolarisation; it is not a pure $\beta$-blocker.

**Final Answer:** $K^+$ channel block prolonging repolarisation (plus $\beta$-block) $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q59

## Q60.

**Solution**

**Concept — Ticagrelor:** Ticagrelor is a directly acting, reversible $P2Y_{12}$ receptor antagonist. **Reasoning:** It binds the platelet $P2Y_{12}$ ADP receptor reversibly and does not need hepatic conversion to an active metabolite, unlike the prodrug clopidogrel. This gives a faster, more consistent antiplatelet effect. **Why the other options are wrong:**

- (B) Irreversible COX acetylation describes aspirin.
- (C) GpIIb/IIIa blockade describes abciximab/tirofiban.
- (D) Antithrombin activation describes heparin.

**Final Answer:** Reversible P2Y$_{12}$ blockade, no activation needed $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q60

## Q61.

### Solution

**Concept — Diltiazem:** Diltiazem is a non-dihydropyridine calcium-channel blocker with both vascular and cardiac actions. **Reasoning:** Besides arterial vasodilation it blocks L-type calcium channels in the SA and AV nodes, slowing conduction and heart rate. This rate-controlling action makes it useful in supraventricular arrhythmias, unlike the vascular-selective amlodipine. **Why the other options are wrong:**

- (A) It does affect cardiac conduction.
- (C) It acts on the arterial side, not selectively on veins.
- (D) It is not an ACE inhibitor.

**Final Answer:** Also slows SA/AV nodal conduction and heart rate $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q61

## Q62.

### Solution

**Concept — Second-generation H$_1$ antihistamine:** Loratadine blocks peripheral H$_1$ receptors with poor CNS penetration. **Reasoning:** Because it crosses the blood-brain barrier poorly, loratadine controls allergic rhinitis and urticaria with little of the sedation seen with first-generation agents such as chlorpheniramine. **Why the other options are wrong:**

- (A) It blocks H$_1$, not H$_2$, receptors.
- (B) It is a non-sedating second-generation agent.
- (D) It acts by receptor blockade, not mast-cell stabilisation.

**Final Answer:** Non-sedating second-generation $H_1$ antagonist $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q62

## Q63.

### Solution

**Concept — Ketorolac:** Ketorolac is a potent non-selective COX-inhibiting NSAID used as a parenteral analgesic. **Reasoning:** It provides strong analgesia comparable to opioids for short-term post-operative pain, but its inhibition of COX-1 brings significant gastrointestinal-bleeding and renal risk, so use is limited to a few days.
**Why the other options are wrong:**

- (A) It is non-selective, so it does carry gastrointestinal risk.
- (B) It inhibits COX peripherally as well, not only centrally.
- (C) It is an NSAID, not an opioid.

**Final Answer:** Potent non-selective COX inhibitor, short-term use only $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q63

## Q64.

### Solution

**Concept — Probenecid (uricosuric):** Probenecid promotes renal excretion of uric acid. **Reasoning:** It inhibits the proximal-tubule urate transporter (URAT1) that normally reabsorbs filtered urate, so more uric acid is excreted and serum urate falls. It is used for chronic gout in under-excretors with good renal function.
**Why the other options are wrong:**

- (B) Xanthine oxidase inhibition describes allopurinol/febuxostat.
- (C) Microtubule inhibition describes colchicine.
- (D) It is not a COX-2 inhibitor.

**Final Answer:** Uricosuric, blocks tubular urate reabsorption $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q64

## Q65.

### Solution

**Concept — Macrolides:** Clarithromycin binds the 50S ribosomal subunit. **Reasoning:** By binding the 23S rRNA of the 50S subunit it blocks translocation of the growing peptide, inhibiting bacterial protein synthesis (a bacteriostatic action). **Why the other options are wrong:**

- (A) 30S binding with misreading describes aminoglycosides.
- (C) DNA gyrase inhibition describes fluoroquinolones.
- (D) Transpeptidase inhibition describes $\beta$-lactams.

**Final Answer:** 50S binding blocking translocation $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q65

## Q66.

### Solution

**Concept — Rifampicin:** Rifampicin inhibits bacterial DNA-dependent RNA polymerase. **Reasoning:** By binding the $\beta$-subunit of RNA polymerase it blocks initiation of RNA transcription, a bactericidal action central to antitubercular therapy. It also induces hepatic enzymes and colours secretions orange-red. **Why the other options are wrong:**

- (A) Mycolic-acid synthesis inhibition describes isoniazid.
- (B) 30S inhibition describes aminoglycosides/tetracyclines.
- (D) DHFR inhibition describes trimethoprim.

**Final Answer:** Inhibits bacterial RNA polymerase (transcription) $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q66

## Q67.

### Solution

**Concept — Acyclovir:** Acyclovir is a guanosine analogue selectively activated in herpesvirus-infected cells. **Reasoning:** The viral thymidine kinase performs the first phosphorylation, so activation occurs preferentially in infected cells; host kinases then form acyclovir triphosphate, which inhibits viral DNA polymerase and, lacking a 3'-OH, terminates the growing DNA chain. This gives its selectivity and low toxicity. **Why the other options are wrong:**

- (A) Neuraminidase inhibition describes oseltamivir.
- (B) It does not block CD4-mediated entry.
- (C) It is selectively activated by viral thymidine kinase, not randomly in all cells, and targets herpesviruses, not HIV.

**Final Answer:** Viral-TK-activated chain terminator of viral DNA polymerase $\Rightarrow \boxed{\text{D}}$

$\boxed{\textbf{Answer: (D)}}$ **Go Back to Q67**

## Q68.

**Solution**

**Concept — Thiazolidinediones:** Pioglitazone is a PPAR-$\gamma$ agonist insulin sensitiser. **Reasoning:** By activating the nuclear receptor PPAR-$\gamma$ it alters transcription of genes regulating glucose and lipid metabolism in adipose, muscle and liver, increasing peripheral insulin sensitivity and lowering blood glucose without directly stimulating insulin release (so monotherapy rarely causes hypoglycaemia). **Why the other options are wrong:**

- (B) Closing $K_{ATP}$ channels to release insulin describes sulfonylureas.
- (C) $\alpha$-glucosidase inhibition describes acarbose.
- (D) SGLT2 inhibition describes the gliflozins.

**Final Answer:** PPAR-$\gamma$ activation enhancing insulin sensitivity $\Rightarrow \boxed{\text{A}}$

$\boxed{\textbf{Answer: (A)}}$ **Go Back to Q68**

## Q69.

**Solution**

**Concept — Rosenmund reduction:** Selective hydrogenation of an acyl chloride to an aldehyde over a poisoned palladium catalyst. **Reasoning:** Pd is deposited on barium sulfate and "poisoned" (e.g. with quinoline/sulfur) so that hydrogenation stops at the aldehyde and does not over-reduce it to the alcohol. This R–COCl $\rightarrow$ R–CHO conversion is the Rosenmund reduction. **Why the other options are wrong:**

- (A) Stephen reduction converts a nitrile (not an acid chloride) to an aldehyde using $SnCl_2$/HCl.

- (C) Clemmensen reduces a carbonyl all the way to a $CH_2$ group (Zn–Hg/HCl).
- (D) Bouveault–Blanc reduces an ester to a primary alcohol (Na/EtOH).

**Final Answer:** Rosenmund reduction ⇒ $\boxed{\text{B}}$

**Answer: (B)** Go Back to Q69

## Q70.

### Solution

**Concept — Vilsmeier–Haack formylation:** Electron-rich aromatics are formylated by a chloromethyleneiminium electrophile. **Reasoning:** $POCl_3$ activates DMF to form the Vilsmeier reagent $[(CH_3)_2N{=}CHCl]^+$, which attacks the activated ring; aqueous work-up hydrolyses the iminium intermediate to the aryl aldehyde. This is the Vilsmeier–Haack reaction. **Why the other options are wrong:**

- (B) Gattermann–Koch uses CO/HCl with $AlCl_3$/CuCl on simple arenes.
- (C) Kolbe–Schmitt carboxylates phenoxide with $CO_2$.
- (D) Houben–Hoesch gives aryl ketones from nitriles, not formylation.

**Final Answer:** Vilsmeier–Haack reaction ⇒ $\boxed{\text{A}}$

**Answer: (A)** Go Back to Q70

## Q71.

### Solution

**Concept — Schmidt reaction:** Acid-catalysed reaction of a carboxylic acid with $HN_3$ giving an amine with loss of one carbon. **Reasoning:** $HN_3$ adds to the protonated acid to form an acyl azide; loss of $N_2$ gives an acyl nitrene that undergoes 1,2-migration to an isocyanate, which is hydrolysed (losing $CO_2$) to a primary amine. This C→N degradation is the Schmidt reaction. **Why the other options are wrong:**

- (A) Favorskii rearranges $\alpha$-halo ketones (ring contraction), no nitrogen.
- (B) Pinacol–pinacolone is a 1,2-shift forming a ketone, no nitrogen.
- (C) Chichibabin aminates pyridine with $NaNH_2$; it adds, not degrades.

**Final Answer:** Schmidt reaction ⇒ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q71

## Q72.

### Solution

**Concept — Pinacol–pinacolone rearrangement:** Acid-catalysed 1,2-shift of a 1,2-diol to a ketone. **Reasoning:** Protonation and loss of water from one –OH gives a carbocation; a 1,2-methyl (or alkyl/aryl) group migrates to it, and loss of a proton from the remaining –OH yields the more stable ketone (pinacolone). The migratory aptitude follows the group's ability to stabilise positive charge. **Why the other options are wrong:**

- (A) "Wagner ring expansion" is not the name for this acyclic 1,2-shift.
- (C) Benzilic acid rearrangement converts a 1,2-diketone to an $\alpha$-hydroxy acid.
- (D) Tiffeneau–Demjanov is a ring expansion of $\beta$-amino alcohols via diazonium.

**Final Answer:** Pinacol–pinacolone rearrangement $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q72

## Q73.

### Solution

**Concept — Mitsunobu reaction:** DEAD + $PPh_3$ couple an alcohol to an acidic pronucleophile with inversion. **Reasoning:** $PPh_3$ and DEAD activate the alcohol as an alkoxyphosphonium species; the nucleophile (e.g. a carboxylate) then displaces it by an $S_N2$ backside attack, inverting the stereocentre. This is the Mitsunobu reaction, widely used to invert chiral secondary alcohols. **Why the other options are wrong:**

- (B) The Appel reaction converts an alcohol to a halide ($CBr_4/PPh_3$), also with inversion, but not the DEAD ester coupling described.
- (C) Corey–House couples a Gilman cuprate with an alkyl halide.
- (D) Tsuji–Trost is a Pd-catalysed allylic substitution.

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

**Answer: (A)** Go Back to Q73

## Q74.

### Solution

**Concept — Chichibabin reaction:** Direct amination of pyridine at C-2 by sodium amide. **Reasoning:** The amide ion (a strong nucleophile) adds to the electron-poor C-2 of pyridine; rearomatisation expels hydride, which combines with H to release $H_2$ and gives 2-aminopyridine. This is the Chichibabin reaction. **Why the other options are wrong:**

- (A) The Bucherer reaction interconverts naphthols and naphthylamines.
- (B) The Skraup synthesis builds the quinoline ring from aniline and glycerol.
- (D) The Combes synthesis builds quinolines from anilines and 1,3-diketones.

**Final Answer:** Chichibabin reaction $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q74

## Q75.

### Solution

**Concept — Nitrogen basicity and lone-pair availability:** Basicity depends on whether the lone pair is free and on the nitrogen's hybridisation. **Reasoning:** In pyrrole the N lone pair is delocalised into the aromatic sextet, so it is essentially non-basic (least basic). In pyridine the lone pair sits in an in-plane $sp^2$ orbital (weak base, $pK_{aH} \approx 5$). In piperidine the saturated $sp^3$ amine has a fully available lone pair (strong base, $pK_{aH} \approx 11$). Increasing order: pyrrole $<$ pyridine $<$ piperidine, i.e. (i) $<$ (ii) $<$ (iii). **Why the other options are wrong:**

- (A), (B) and (D) all misplace pyrrole or invert the pyridine/piperidine ranking.
- Only (i) $<$ (ii) $<$ (iii) reflects the true availability of the lone pair.

**Final Answer:** (i) $<$ (ii) $<$ (iii) $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q75

## Q76.

### Solution

**Concept — Favorskii rearrangement:** Base converts an $\alpha$-halo ketone to a ring-contracted ester via a cyclopropanone. **Reasoning:** The alkoxide removes the $\alpha'$-proton; the resulting enolate displaces the halide intramolecularly to form a strained cyclopropanone, which the alkoxide opens to give the ring-contracted carboxylic ester. With a cyclic $\alpha$-halo ketone this gives a smaller ring acid/ester. **Why the other options are wrong:**

- (A) Neber rearrangement converts oxime tosylates to $\alpha$-amino ketones.
- (C) Meinwald rearranges epoxides to carbonyls.
- (D) Ramberg–Bäcklund extrudes $SO_2$ from $\alpha$-halo sulfones to give alkenes.

**Final Answer:** Favorskii rearrangement $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q76

## Q77.

### Solution

**Concept — Stephen reaction:** Partial reduction of a nitrile to an aldehyde. **Reasoning:** Anhydrous $SnCl_2$ with HCl reduces R–C≡N to an aldimine hydrochloride (R–CH=NH·HCl); aqueous hydrolysis then liberates the aldehyde R–CHO. This is the Stephen reduction. **Why the other options are wrong:**

- (B) The Etard reaction oxidises toluene's methyl to –CHO with $CrO_2Cl_2$.
- (C) The Gattermann reaction formylates arenes (HCN/HCl), a different transform.
- (D) The Sonn–Müller method makes aldehydes from anilides via imidoyl chlorides, not from nitriles with $SnCl_2$.

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

**Answer: (A)** Go Back to Q77

## Q78.

**Solution**

**Concept — Nucleophilicity in protic solvents:** Larger, more polarisable halides are less tightly solvated and thus better nucleophiles in protic media. **Reasoning:**

In methanol, hydrogen bonding solvates the small $F^-$ very strongly, hindering it; the larger $I^-$ is poorly solvated and most polarisable, so nucleophilicity rises down the group: $I^- > Br^- > Cl^- > F^-$, i.e. (iv) > (iii) > (ii) > (i). **Why the other options are wrong:**

- (A) Gives the gas-phase/basicity order, opposite to the protic-solvent trend.
- (C) and (D) scramble the halide ranking.

**Final Answer:** (iv) > (iii) > (ii) > (i) $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q78

## Q79.

**Solution**

**Concept — Thiazole:** A 1,3-azole containing one sulfur (position 1) and one nitrogen (position 3). **Reasoning:** The five-membered aromatic ring with S at 1

and N at 3 is thiazole. Its ring is the core of thiamine (vitamin $B_1$) and is fused into the penicillin nucleus. The pyridine-type N makes it weakly basic; the ring is aromatic (6 $\pi$ electrons). **Why the other options are wrong:**

- (A) Oxazole has oxygen (not sulfur) at position 1.
- (B) Isoxazole has adjacent O and N (1,2-positions).
- (C) Imidazole has two nitrogens (1,3) and no sulfur.

**Final Answer:** The heterocycle is thiazole $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q79

## Q80.

**Solution**

**Concept — Oxymercuration mechanism:** Markovnikov hydration that avoids carbocation rearrangement. **Reasoning:** Mercury(II) forms a bridged *mer-*

*curinium* ion across the double bond; water opens it at the more substituted carbon (Markovnikov), and demercuration with $NaBH_4$ gives the alcohol. Because no free, open carbocation forms, the neopentyl-type skeleton does not rearrange. **Why the other options are wrong:**

- (A) The product is the Markovnikov alcohol, not anti-Markovnikov.
- (B) The whole point is that no open primary carbocation is involved.
- (D) The bridged ion gives anti addition, but the rearrangement-free outcome is what distinguishes it from acid hydration.

**Final Answer:** Bridged mercurinium ion, no rearrangement $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q80

## Q81.

### Solution

**Concept — Enolate resonance:** The charge is shared between the $\alpha$-carbon and the oxygen. **Reasoning:** The two contributors (carbanion form and the oxyanion/alkene form) are *not* equivalent. Because oxygen is more electronegative, the structure carrying the negative charge on oxygen is the lower-energy, larger contributor, so most of the charge density sits on oxygen, though carbon is the more nucleophilic site. **Why the other options are wrong:**

- (A) The charge is delocalised, not fixed on carbon.
- (B) Resonance delocalisation does occur (no aromaticity is required).
- (C) The two contributors are inequivalent, so they do not contribute equally.

**Final Answer:** Charge delocalised on O and C; O form dominates $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q81

## Q82.

### Solution

**Concept — Bouveault–Blanc reduction:** Dissolving-metal reduction of esters to primary alcohols. **Reasoning:** Sodium in absolute ethanol supplies single electrons (and the alcohol supplies protons) to reduce R–COOR′ to R–CH$_2$OH plus the alcohol R′OH. It was the standard ester reduction before LiAlH$_4$ and catalytic hydrogenation. **Why the other options are wrong:**

- (B) Meerwein–Ponndorf–Verley reduces ketones/aldehydes to alcohols (Al alkoxide), not esters.

- (C) Rosenmund reduces acid chlorides to aldehydes.
- (D) The Luche reduction is selective 1,2-reduction of enones with $NaBH_4/CeCl_3$.

**Final Answer:** Bouveault–Blanc reduction ⇒ $\boxed{\text{A}}$

$\boxed{\text{Answer: (A)}}$ Go Back to Q82

## Q83.

### Solution

**Concept — Anomers:** Diastereomers differing only in configuration at the new stereocentre formed on ring closure (the anomeric carbon). **Reasoning:** On cyclisation the carbonyl carbon (C-1 in glucose) becomes a hemiacetal stereocentre; the $\alpha$ and $\beta$ forms differ only there, so they are anomers (a special type of epimer/diastereomer). Interconversion in solution is mutarotation. **Why the other options are wrong:**

- (B) Enantiomers are non-superimposable mirror images at *all* centres; here only C-1 differs.
- (C) Conformers interconvert by bond rotation without breaking bonds.
- (D) Constitutional isomers differ in connectivity, not configuration at one carbon.

**Final Answer:** They are anomers ⇒ $\boxed{\text{A}}$

$\boxed{\text{Answer: (A)}}$ Go Back to Q83

## Q84.

### Solution

**Concept — Functional-group recognition:** A carbon triple-bonded to nitrogen is a nitrile. **Reasoning:** The skeletal structure shows a terminal carbon joined to N by a triple bond ($-C\equiv N$), the cyano/nitrile functional group. **Why the other options are wrong:**

- (A) An amide has a $C=O$ bonded to N, not a $C\equiv N$ triple bond.
- (C) An amine has a single C–N bond to nitrogen.
- (D) An imine has a $C=N$ double bond, not a triple bond.

**Final Answer:** Nitrile ($-C\equiv N$) ⇒ $\boxed{\text{B}}$

Answer: (B) Go Back to Q84

## Q85.

### Solution

**Concept — $\beta$-blockers:** The “-olol” aryloxypropanolamines competitively block $\beta$-adrenoceptors. **Reasoning:** Propranolol, atenolol and metoprolol share the Ar–O–$CH_2$–CH(OH)–$CH_2$–NH–R pharmacophore and antagonise $\beta_1/\beta_2$ receptors, reducing heart rate and blood pressure. They are $\beta$-adrenergic receptor antagonists. **Why the other options are wrong:**

- (A) Calcium-channel blockers (e.g. amlodipine) act on $Ca^{2+}$ channels.
- (B) ACE inhibitors are the “-pril” drugs.
- (C) $H_2$ antagonists (e.g. ranitidine) block gastric histamine receptors.

**Final Answer:** $\beta$-adrenergic receptor antagonists $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q85

## Q86.

### Solution

**Concept — Isoniazid mechanism:** A prodrug activated by KatG that blocks mycolic-acid synthesis. **Reasoning:** The mycobacterial catalase-peroxidase KatG oxidatively activates isoniazid; the activated species inhibits InhA (enoyl-ACP reductase), halting biosynthesis of mycolic acids essential to the mycobacterial cell wall. **Why the other options are wrong:**

- (B) Peptidoglycan cross-linking is the $\beta$-lactam/glycopeptide target.
- (C) The 30S ribosome is the aminoglycoside/tetracycline target.
- (D) Dihydrofolate is targeted by antifolates such as trimethoprim.

**Final Answer:** Inhibits mycolic-acid synthesis $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q86

## Q87.

**Solution**

**Concept — Nitrogen-mustard alkylating agents:** They covalently cross-link DNA via an aziridinium electrophile. **Reasoning:** Cyclophosphamide (after hepatic activation) and mechlorethamine cyclise to a reactive aziridinium ion that alkylates N-7 of guanine; the second arm alkylates a guanine on the opposite strand, giving interstrand cross-links that block replication. They are bifunctional alkylating agents. **Why the other options are wrong:**

- (A) Antimetabolites (e.g. methotrexate) mimic normal metabolites; no covalent DNA cross-link.
- (B) Topoisomerase inhibitors trap the enzyme–DNA complex.
- (D) Tubulin binders (e.g. vinca alkaloids) disrupt the mitotic spindle.

**Final Answer:** Bifunctional alkylating agents $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q87

## Q88.

**Solution**

**Concept — Partial agonist:** Binds the receptor but gives a sub-maximal ceiling response. **Reasoning:** A partial agonist has intrinsic efficacy between 0 and that of a full agonist, so it cannot evoke the maximal effect even at full occupancy. In the presence of a full agonist it competes for the same site and lowers the overall response, behaving as a partial antagonist. **Why the other options are wrong:**

- (A) A full agonist produces the maximal response.
- (C) A neutral antagonist has zero efficacy (no response of its own).
- (D) An inverse agonist reduces constitutive (baseline) receptor activity below zero.

**Final Answer:** Partial agonist $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q88

## Q89.

**Solution**

**Concept — Drug-receptor binding forces:** Among reversible interactions, the ionic bond is the strongest. **Reasoning:** A protonated amine (–$NH_3^+$) and an ionised carboxylate/phosphate form an electrostatic (ionic) attraction, the strongest of the non-covalent forces (typically 20–40 $\mathrm{kJ\,mol^{-1}}$), giving the initial long-range "anchor" for the ligand. **Why the other options are wrong:**

- (B) Van der Waals/London forces are weak and very short-range.
- (C) Hydrophobic interactions are entropy-driven and individually weak.
- (D) Dipole–dipole interactions are weaker than a full ionic interaction.

**Final Answer:** Ionic (electrostatic) interaction $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** **Go Back to Q89**

## Q90.

**Solution**

**Concept — Chiral (racemic) switch:** Re-marketing a single enantiomer of a previously racemic drug. **Reasoning:** Esomeprazole (*S*-omeprazole) and levocetirizine (*R*-cetirizine) are single-enantiomer versions of earlier racemates, developed for cleaner pharmacokinetics or fewer side effects. This life-cycle strategy is a chiral (racemic) switch. **Why the other options are wrong:**

- (A) Bioisosterism replaces a group, not an enantiomer.
- (B) Scaffold hopping changes the core ring system.
- (C) A prodrug is a metabolically activated derivative, not an enantiomer switch.

**Final Answer:** A chiral (racemic) switch $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** **Go Back to Q90**

## Q91.

**Solution**

**Concept — Warfarin mechanism:** 4-Hydroxycoumarin anticoagulants block vitamin-K recycling. **Reasoning:** Warfarin inhibits vitamin K epoxide reductase (VKORC1), preventing regeneration of reduced vitamin K needed for $\gamma$-

carboxylation of glutamate residues in factors II, VII, IX and X, so functional clotting factors are not made. **Why the other options are wrong:**

- (A) COX is the NSAID/aspirin target.
- (C) Direct thrombin inhibition is the action of dabigatran, not warfarin.
- (D) HMG-CoA reductase is the statin target.

**Final Answer:** Vitamin K epoxide reductase (VKORC1) $\Rightarrow \boxed{\text{B}}$

$\boxed{\textbf{Answer: (B)}}$ **Go Back to Q91**

## Q92.

**Solution**

**Concept — Vancomycin target:** It binds the D-Ala–D-Ala terminus of the peptidoglycan precursor. **Reasoning:** Vancomycin forms five hydrogen bonds to the terminal D-alanyl-D-alanine of lipid-II/pentapeptide, sterically blocking transglycosylation and transpeptidation. Resistance arises when the terminus mutates to D-Ala–D-Lac, weakening binding. **Why the other options are wrong:**

- (A) The 50S subunit is the macrolide target.
- (B) DNA gyrase is the fluoroquinolone target.
- (D) Dihydropteroate synthase is the sulfonamide target.

**Final Answer:** The terminal D-Ala–D-Ala $\Rightarrow \boxed{\text{C}}$

$\boxed{\textbf{Answer: (C)}}$ **Go Back to Q92**

## Q93.

**Solution**

**Concept — Apparent zero-order kinetics:** When the dissolved concentration is held constant, the rate is constant. **Reasoning:** In a suspension the dissolved drug is constantly replenished from undissolved solid, so $[\text{drug in solution}]$ stays at the saturation solubility. The degradation rate, being proportional to this fixed concentration, is constant with time, giving apparent (pseudo) zero-order loss until the solid reservoir is exhausted. **Why the other options are wrong:**

- (A) True first order would require the dissolved concentration to fall continuously.
- (B) and (D) Second/fractional orders do not fit a constant-concentration

reservoir.

**Final Answer:** Apparent zero order $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q93

## Q94.

### Solution

**Concept — Arrhenius equation:** Temperature mainly affects the Boltzmann factor $e^{-E_a/RT}$. **Reasoning:** A modest rise in $T$ raises the fraction of molecules with energy $\geq E_a$ exponentially, which is why rate constants can roughly double for a $10\,^{\circ}$C rise. The average kinetic energy itself rises only slightly; it is the tail of the distribution above $E_a$ that grows sharply. **Why the other options are wrong:**

- (B) The average energy increases only modestly, far less than the rate.
- (C) $E_a$ is essentially temperature-independent for a given reaction.
- (D) $\Delta H$ is a thermodynamic state function, unrelated to the rate constant.

**Final Answer:** Exponential rise in the fraction with $E \geq E_a \Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q94

## Q95.

### Solution

**Concept — Sodium bicarbonate:** A systemic (absorbable) antacid and urinary alkaliniser. **Reasoning:** $NaHCO_3$ neutralises gastric HCl rapidly; because it is absorbed, excess use raises systemic pH (metabolic alkalosis) and alkalinises the urine, which is exploited to speed renal elimination of weakly acidic drugs (e.g. salicylate, barbiturate overdose). **Why the other options are wrong:**

- (A) Calcium carbonate is largely non-systemic and can cause acid rebound.
- (C) Aluminium hydroxide is a non-systemic antacid (and constipating).
- (D) Magnesium trisilicate is a non-absorbable adsorbent antacid.

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

**Answer: (B)** Go Back to Q95

## Q96.

### Solution

**Concept — Wacker process:** Pd(II)/Cu(II)-catalysed aerobic oxidation of a terminal alkene to a methyl ketone (or, for ethene, to acetaldehyde). **Reasoning:** Water

attacks a Pd(II)–alkene complex; $\beta$-hydride elimination/reductive steps give the carbonyl while Pd(0) is reoxidised to Pd(II) by Cu(II), which $O_2$ then re-oxidises. For ethene the product is acetaldehyde. This is the Wacker (Wacker–Tsuji) process. **Why the other options are wrong:**

- (A) The Heck reaction couples an aryl/vinyl halide with an alkene (no carbonyl formed).
- (B) Oppenauer oxidises secondary alcohols to ketones (Al alkoxide), not alkenes.
- (D) The Etard reaction oxidises a methyl arene to an aldehyde with $CrO_2Cl_2$.

**Final Answer:** Wacker process $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q96

## Q97.

### Solution

**Concept — 1,3-Diaxial interactions:** Steric strain between an axial substituent and the axial H’s at C-3 and C-5. **Reasoning:** When the methyl is axial it crowds

the two axial hydrogens on the same face of the ring (at C-3 and C-5), raising the energy. Placing the methyl equatorial relieves these 1,3-diaxial interactions, which is why the equatorial chair predominates. **Why the other options are wrong:**

- (A) Torsional strain arises from eclipsing bonds, not 1,3-crowding.
- (B) Angle (Baeyer) strain is bond-angle distortion, negligible in a chair.
- (C) Gauche butane strain describes the related interaction along one bond, but the specific term for both axial H clashes is 1,3-diaxial interaction.

**Final Answer:** 1,3-Diaxial interactions $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q97

## Q98.

### Solution

**Concept — Intermediates vs transition states:** A species in an energy *valley* is an intermediate; a species at a *maximum* is a transition state. **Reasoning:** Point X sits in the well between $TS_1$ and $TS_2$, so it is a reaction intermediate (a real, if short-lived, species). The rate-determining step is the one crossing the highest-energy transition state; here $TS_1$ is higher than $TS_2$, so the first step is rate-determining. **Why the other options are wrong:**

- (A) and (B) X is not a transition state (it is an energy minimum, not a maximum).
- (C) The rate-determining step is governed by $TS_1$ (the higher barrier), not $TS_2$.

**Final Answer:** X is an intermediate; step 1 ($TS_1$) is rate-determining $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q98

## Q99.

### Solution

**Concept — Beer–Lambert law:** $A = \varepsilon c l$, so a plot of $A$ versus $c$ has slope $\varepsilon l$. **Reasoning:** The fitted slope is $\varepsilon l = 1.20 \times 10^3$ (with $c$ in mol $L^{-1}$). Since $l = 1.0$ cm, $\varepsilon = (\text{slope})/l = 1.20 \times 10^3$ L mol$^{-1}$ cm$^{-1}$. Note molar absorptivity carries units L mol$^{-1}$ cm$^{-1}$. **Why the other options are wrong:**

- (A) Has the units inverted (mol L$^{-1}$ cm$^{-1}$ is not the unit of $\varepsilon$).
- (B) Is the reciprocal of the slope, the wrong magnitude.
- (D) Drops the $10^3$ factor entirely.

**Final Answer:** $\varepsilon = 1.20 \times 10^3$ L mol$^{-1}$ cm$^{-1}$ $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q99

## Q100.

### Solution

**Concept — Normality and electron change:** $N = M \times$ (electrons exchanged per mole) for a redox titrant. **Reasoning:** Under strong acid the bromate ion gains 6 electrons (Br goes from $+5$ to $-1$). Hence the equivalence factor is 6 and $N = 0.0250 \times 6 = 0.150$ N. **Why the other options are wrong:**

- (B) 0.0250 N treats the change as a one-electron process.
- (C) 0.0500 N uses a factor of 2.
- (D) 0.00417 N divides by 6 instead of multiplying.

**Final Answer:** $N = 0.0250 \times 6 = 0.150\ \text{N} \Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q100

## Q101.

### Solution

**Concept — Karl Fischer endpoint:** excess iodine is detected electrochemically. **Reasoning:** The endpoint is signalled by the first slight excess of iodine, which is sensed by a pair of platinum electrodes held at a small fixed potential. The sudden flow (or cessation) of current marks the end; this is the dead-stop (biamperometric) technique. **Why the other options are wrong:**

- (A) Phenolphthalein is an acid–base indicator, irrelevant to a moisture titration.
- (C) Gravimetric drying is a separate, non-titrimetric method.
- (D) Refractive index is not used to locate a KF endpoint.

**Final Answer:** Dead-stop (biamperometric) endpoint detection $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q101

## Q102.

### Solution

**Concept — Complexometric titration modes:** direct, back, masking, and displacement. **Reasoning:** Adding the Mg–EDTA complex lets the analyte ion (which binds EDTA more strongly) displace an equivalent quantity of $Mg^{2+}$. The released $Mg^{2+}$ is then titrated, giving a sharp endpoint with Eriochrome Black T. This is a displacement (substitution) titration. **Why the other options are wrong:**

- (A) A direct titration adds EDTA straight to the analyte; that fails here for want of a sharp endpoint.
- (B) A back titration adds excess EDTA and titrates the surplus, not the case described.

- (C) Masking suppresses an interferent; it is not the titration mode itself.

**Final Answer:** Displacement (substitution) titration ⇒ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q102

## Q103.

### Solution

**Concept — IR aldehyde C–H:** the aldehydic C–H stretch gives a tell-tale weak doublet near 2720 and 2820 $\text{cm}^{-1}$. **Reasoning:** The two weak bands arise from

Fermi resonance between the aldehyde C–H stretch and the overtone of the C–H bend. Together with a strong C=O band near 1720 $\text{cm}^{-1}$, this doublet confirms an aldehyde (–CHO) group. **Why the other options are wrong:**

- (A) A tertiary amine has no N–H and no such doublet.
- (B) An ether shows a strong C–O–C stretch near 1100 $\text{cm}^{-1}$, not this doublet.
- (D) A sulphonic acid shows strong S=O bands near 1350 and 1150 $\text{cm}^{-1}$.

**Final Answer:** 2720/2820 $\text{cm}^{-1}$ doublet ⇒ aldehyde ⇒ $\boxed{\text{C}}$

**Answer: (C)** Go Back to Q103

## Q104.

### Solution

**Concept — Andrews iodate titration:** in concentrated HCl the iodine is stabilised as iodine monochloride (ICl), i.e. iodine in the +1 state. **Reasoning:** The titrant

$IO_3^-$ (iodine +5) is reduced only to ICl (iodine +1), a change of 4 electrons, not all the way to $I^-$. The endpoint (disappearance of the $I_2$/chloroform colour) corresponds to all iodine being converted to ICl. **Why the other options are wrong:**

- (A) Reduction to $I^-$ (6 electrons) applies in dilute, not concentrated HCl conditions.
- (B) $IO_3^- \rightarrow I_2$ is not the Andrews endpoint state.
- (C) Iodate is being reduced here, not oxidised to periodate.

**Final Answer:** $IO_3^- \rightarrow$ ICl, 4-electron change ⇒ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q104

## Q105.

### Solution

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

- (B) 2.50 is the inverse ratio (12/4.8); R$_f$ cannot exceed 1.
- (C) 0.48 misplaces the decimal point.
- (D) 0.60 uses a wrong solute distance (7.2 cm).

**Final Answer:** $R_f = 4.8/12.0 = 0.40 \Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q105

## Q106.

### Solution

**Concept — Column efficiency:** using baseline (tangent) width, $N = 16\,(t_R/w)^2$. **Reasoning:** $t_R/w = 6.0/0.30 = 20$, so $N = 16 \times 20^2 = 16 \times 400 = 6400$ theoretical plates. **Why the other options are wrong:**

- (A) 320 forgets to square the ratio.
- (B) 1600 uses the half-height factor 5.54 instead of 16, or a wrong width.
- (D) 400 is $(t_R/w)^2$ without the factor 16.

**Final Answer:** $N = 16(20)^2 = 6400 \Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q106

## Q107.

### Solution

**Concept — Fluorescence intensity:** $F = k\,\Phi I_0 \varepsilon c l$, which is linear in $c$ only for low absorbance. **Reasoning:** The proportionality $F \propto c$ holds when the solution is dilute (absorbance roughly $< 0.05$). At higher concentrations the inner-filter effect and self-absorption bend the calibration, so linearity is lost. **Why the other options are wrong:**

- (A) Concentrated, strongly absorbing solutions are exactly where linearity breaks down.

- (C) Excitation and emission wavelengths differ by the Stokes shift; equality is not the condition.
- (D) Zero quantum yield means no fluorescence at all.

**Final Answer:** Linear only in dilute, low-absorbance solutions $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q107

## Q108.

### Solution

**Concept — Selectivity factor:** $\alpha = t'_{R2}/t'_{R1}$ (ratio of adjusted retention times, $\alpha \geq 1$). **Reasoning:** With $t'_{R1} = 2.0$ min and $t'_{R2} = 3.0$ min, $\alpha = 3.0/2.0 = 1.5$. A value greater than 1 means the two solutes are differentially retained. **Why the other options are wrong:**

- (A) 0.67 is the inverse ratio; $\alpha$ is defined $\geq 1$.
- (B) 1.0 would mean no separation at all.
- (C) 5.0 multiplies the two values instead of dividing.

**Final Answer:** $\alpha = 3.0/2.0 = 1.5 \Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q108

## Q109.

### Solution

**Concept — Aromatic ring current:** the circulating $\pi$ electrons of benzene deshield its protons. **Reasoning:** The induced ring current creates a magnetic field that adds to the applied field at the ring protons, deshielding them. Consequently aromatic C–H protons resonate downfield, typically near $\delta$ 7–8 ppm. **Why the other options are wrong:**

- (B) $\delta$ 0–0.5 ppm is upfield of TMS, seen only for very shielded protons.
- (C) $\delta$ 2–2.5 ppm is the region of simple $CH_3/CH_2$ groups, not aromatic H.
- (D) $\delta$ 12–13 ppm is the carboxylic-acid OH region, far downfield of aromatic H.

**Final Answer:** Aromatic protons appear near $\delta$ 7–8 ppm $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q109

## Q110.

**Solution**

**Concept — Equivalence-point pH:** a weak acid–strong base titration yields the salt of a weak acid at equivalence. **Reasoning:** At point E all the weak acid has been converted to its conjugate base (e.g. acetate). This anion hydrolyses water to give $OH^-$, so the equivalence-point pH is above 7 (basic). This is why the inflection sits higher than 7 on the curve. **Why the other options are wrong:**

- (A) pH 7 is the equivalence point only for a strong acid–strong base pair.
- (B) No excess acid remains exactly at equivalence; the solution is not acidic.
- (D) The equivalence pH clearly depends on the acid's strength ($pK_a$).

**Final Answer:** Equivalence-point pH is above 7 (basic) $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q110

## Q111.

**Solution**

**Concept — Bromine isotope pattern:** bromine has two abundant isotopes in almost equal amounts. **Reasoning:** $^{79}$Br (≈50.7%) and $^{81}$Br (≈49.3%) occur in nearly 1:1 abundance, so a single-Br compound gives M and M+2 peaks of almost equal height, separated by 2 mass units. **Why the other options are wrong:**

- (A) Bromine is not monoisotopic; an M+2 peak does appear.
- (C) $^{35}/^{37}$ in 3:1 is the chlorine pattern, not bromine.
- (D) Bromine has two, not three, common isotopes; there is no large M+1 here.

**Final Answer:** $^{79}$Br and $^{81}$Br in ≈1:1 abundance $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q111

## Q112.

**Solution**

**Concept — AAS components:** source (hollow-cathode lamp), atomiser (flame), monochromator, detector. **Reasoning:** The nebuliser/burner sprays the sample into the flame, where the solvent evaporates and the analyte is desolvated, vaporised, and dissociated into free ground-state atoms. These atoms then absorb the resonance-line radiation, the basis of AAS. **Why the other options are wrong:**

- (A) Dispersing the radiation is the monochromator's job.
- (B) The resonance line is produced by the hollow-cathode lamp.
- (C) Detection and amplification are done by the photomultiplier/detector.

**Final Answer:** The atomiser produces free ground-state atoms $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q112

## Q113.

### Solution

**Concept — Kovats retention index:** a standardised retention scale referenced to $n$-alkanes. **Reasoning:** Each $n$-alkane is assigned an index of 100 times its carbon number (hexane = 600, heptane = 700, etc.). A solute's index is obtained by logarithmic interpolation of its adjusted retention between the bracketing $n$-alkanes, giving a near-instrument-independent identifier. **Why the other options are wrong:**

- (B) The reference series is $n$-alkanes, not primary alcohols.
- (C) Aromatics are not the index reference and are not ranked alphabetically.
- (D) Fatty-acid methyl esters are analytes, not the Kovats reference scale.

**Final Answer:** Referenced to $n$-alkanes (index $= 100\times$ carbon number) $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q113

## Q114.

### Solution

**Concept — 1:1 EDTA stoichiometry:** mmol metal $=$ mmol EDTA $= M \times V$(mL). **Reasoning:** mmol EDTA $= 0.0100 \times 25.0 = 0.250$ mmol. Because EDTA binds $Ca^{2+}$ in a 1:1 ratio, the sample contains 0.250 mmol of $Ca^{2+}$. **Why the other options are wrong:**

- (A) 0.500 mmol doubles the value (wrong 1:2 stoichiometry).
- (B) 2.50 mmol misplaces the decimal by ten.
- (D) 0.0250 mmol misplaces the decimal by ten the other way.

**Final Answer:** $0.0100 \times 25.0 = 0.250$ mmol $Ca^{2+} \Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q114

## Q115.

**Solution**

**Concept — Chromophore:** the covalently unsaturated group responsible for UV/visible absorption. **Reasoning:** A chromophore (e.g. C=O, C=C, an aromatic ring, –N=N–) contains the $\pi$ or $n$ electrons whose excitation produces the absorption band marked at $\lambda_{max}$. It is the light-absorbing functional unit of the molecule. **Why the other options are wrong:**

- (A) An auxochrome (e.g. –OH, $-NH_2$) does not absorb on its own; it shifts/intensifies the chromophore's band.
- (C) "Bathochrome" is not the name of the absorbing group; a bathochromic shift is a red shift.
- (D) A fluorophore quencher reduces fluorescence; it is not the UV-absorbing group here.

**Final Answer:** The absorbing unsaturated group is a chromophore $\Rightarrow \boxed{B}$

**Answer: (B)** Go Back to Q115

## Q116.

**Solution**

**Concept — HPLC flow path:** reservoir → pump → injector → column → detector. **Reasoning:** The component at the very start, holding the mobile-phase solvent before the pump draws it in, is the mobile-phase (solvent) reservoir. Everything downstream depends on solvent being supplied from here. **Why the other options are wrong:**

- (A) The detector sits at the end of the flow path, after the column.
- (B) The column lies between injector and detector, not at the start.
- (C) The injector is between pump and column, not before the pump.

**Final Answer:** R is the mobile-phase (solvent) reservoir $\Rightarrow \boxed{D}$

**Answer: (D)** Go Back to Q116

## Q117.

### Solution

**Concept — Indicator selection:** the indicator's transition range must straddle the equivalence-point pH. **Reasoning:** A weak acid–strong base titration has its equivalence point in the basic region. Phenolphthalein changes from colourless to pink over about pH 8.0–10.0, matching that jump, so it is the indicator of choice. **Why the other options are wrong:**

- (B) pH 3.1–4.4 (red to yellow) describes methyl orange, suited to strong acid–weak base.
- (C) pH 0–2 lies far below any normal equivalence point.
- (D) pH 12–14 is too high; phenolphthalein actually fades back to colourless above ≈13, and its useful change is 8–10.

**Final Answer:** Phenolphthalein changes over pH $\approx$ 8–10 $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q117

## Q118.

### Solution

**Concept — Flame photometry:** each alkali metal emits at a characteristic wavelength. **Reasoning:** Sodium emits its intense yellow doublet (the D lines) at about 589 nm when excited atoms relax to the ground state. This emission is measured for quantitative sodium estimation. **Why the other options are wrong:**

- (A) ≈422 nm is a calcium emission, not sodium.
- (C) ≈766 nm is the potassium line.
- (D) 254 nm is a mercury UV line, irrelevant to sodium flame emission.

**Final Answer:** Sodium D lines at ≈589 nm $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q118

## Q119.

### Solution

**Concept — SEC elution window:** all separation occurs between $V_0$ (excluded) and $V_t$ (total permeation). **Reasoning:** The largest molecules are excluded from the pores and elute first at $V_0$; the smallest fully permeate and elute last at $V_t$.

Intermediate-size molecules partially enter the pores and elute somewhere between $V_0$ and $V_t$, with $V_e$ increasing as the molecule gets smaller. **Why the other options are wrong:**

- (A) Nothing elutes before $V_0$; that is the earliest possible volume.
- (B) Only fully excluded species elute exactly at $V_0$.
- (D) Nothing elutes after $V_t$ in ideal SEC; that is the latest volume.

**Final Answer:** $V_e$ lies between $V_0$ and $V_t$, larger for smaller molecules $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q119

## Q120.

### Solution

**Concept — TMS as NMR reference:** an inert, symmetric, volatile standard giving one sharp upfield line. **Reasoning:** TMS, $Si(CH_3)_4$, has 12 chemically equivalent, highly shielded protons that give a single sharp peak at higher field than almost all organic protons. It is chemically inert and easily removed by evaporation, so it is set as $\delta = 0$. **Why the other options are wrong:**

- (A) UV absorption at 254 nm is irrelevant to an NMR reference.
- (B) TMS has no unpaired electron; that would describe an EPR/radical species.
- (C) TMS resonates upfield (not downfield) of nearly all organic protons.

**Final Answer:** Inert, volatile, 12 equivalent protons giving one sharp upfield peak $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** Go Back to Q120

## Q121.

### Solution

**Concept — Coulometry:** the analyte amount follows from the charge passed via Faraday's laws. **Reasoning:** At 100% current efficiency, the total charge $Q$ (coulombs) equals $nFN$, where $n$ is the electrons per molecule, $F$ the Faraday constant, and $N$ the moles reacted. Measuring $Q$ therefore gives $N$ directly, with no standard solution needed. **Why the other options are wrong:**

- (B) Beer's law relates absorbance to concentration in spectrophotometry, not charge.

- (C) Henderson–Hasselbalch deals with buffer pH, not coulometry.
- (D) The van Deemter equation describes chromatographic band-broadening.

**Final Answer:** Faraday's laws of electrolysis ($Q = nFN$) $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q121

## Q122.

**Solution**

**Concept — ICH stability testing:** accelerated conditions stress the product to predict shelf life. **Reasoning:** For a typical solid oral product stored at room temperature, the standard accelerated condition is $40\,°\text{C} \pm 2\,°\text{C}$ and 75% $\pm 5\%$ relative humidity for 6 months. A significant change at these conditions triggers intermediate-condition testing. **Why the other options are wrong:**

- (A) $5\,°$C is a refrigerated long-term condition, not accelerated.
- (C) $-20\,°$C is a freezer condition, used for special products, not the accelerated stress.
- (D) $60\,°$C/0% RH is a forced-degradation (stress) condition, not the defined accelerated stability point.

**Final Answer:** $40\,°$C/75% RH for 6 months $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** Go Back to Q122

## Q123.

**Solution**

**Concept — Alphabetical classification:** Crude drugs may be listed simply in alphabetical order of their Latin or English names, a purely indexing arrangement used in pharmacopoeias and dictionaries. **Reasoning:** The sequence *Acacia*, *Belladonna*, *Cinchona*, *Digitalis*, *Ergota* follows the letters A, B, C, D, E with no regard to botany, chemistry or therapeutics, which is alphabetical classification. **Why the other options are wrong:**

- (A) Morphological grouping is by plant part used.
- (C) Chemical grouping is by constituent class.
- (D) Serological grouping is by genetic/immunological relationship, not used here.

**Final Answer:** An A–Z name listing is alphabetical classification ⇒ $\boxed{\text{B}}$

Answer: (B) Go Back to Q123

## Q124.

### Solution

**Concept — Taxonomical classification:** Crude drugs are arranged according to their botanical (taxonomic) position, that is by phylum, order, family, genus and species. **Reasoning:** Placing *Belladonna*, *Stramonium* and *Hyoscyamus* together solely because they belong to the family Solanaceae groups them by botanical relationship, which is taxonomical classification. **Why the other options are wrong:**

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

**Final Answer:** Grouping by botanical family is taxonomical classification ⇒ $\boxed{\text{C}}$

Answer: (C) Go Back to Q124

## Q125.

### Solution

**Concept — Morphological (organized vs unorganized):** Organized drugs are whole plant organs (leaves, barks, roots, seeds) with a cellular structure; unorganized drugs are cell-free exudates such as gums, resins, balsams and dried extracts. **Reasoning:** Buchu is the dried leaf of *Barosma betulina*, a genuine organized leaf drug with cellular tissue, whereas the other three are cell-free products. **Why the other options are wrong:**

- (A) Tragacanth is a dried gummy exudate (unorganized).
- (B) Benzoin is a balsamic resin (unorganized).
- (C) Catechu is a dried aqueous extract (unorganized).

**Final Answer:** Buchu leaf is the organized drug ⇒ $\boxed{\text{D}}$

Answer: (D) Go Back to Q125

## Q126.

### Solution

**Concept — Yohimbine:** Yohimbine is an indole alkaloid related to the corynanthe/ajmalicine series. **Reasoning:** It is obtained chiefly from the bark of *Pausinystalia yohimbe* (formerly *Corynanthe yohimbe*) and acts as a peripheral $\alpha_2$-adrenoceptor antagonist with aphrodisiac use. **Why the other options are wrong:**

- (B) *Erythroxylum coca* yields cocaine.
- (C) *Papaver somniferum* yields morphine/codeine.
- (D) *Claviceps purpurea* yields ergot alkaloids.

**Final Answer:** Source is *Pausinystalia yohimbe* bark $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** Go Back to Q126

## Q127.

### Solution

**Concept — Ergot alkaloids:** Ergot is the dried sclerotium of the fungus *Claviceps purpurea* developed on the ovary of rye, *Secale cereale*. **Reasoning:** Ergotamine is a peptide-type ergoline alkaloid of ergot, used as an oxytocic and in the treatment of migraine. The drug described is therefore ergot. **Why the other options are wrong:**

- (A) Opium is the poppy latex (morphine group).
- (B) Vinca yields vinblastine/vincristine.
- (D) Ipecacuanha yields emetine.

**Final Answer:** The drug is ergot of *Claviceps purpurea* $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** Go Back to Q127

## Q128.

### Solution

**Concept — Cocaine ester hydrolysis:** Cocaine is the methyl ester of benzoylecgonine, i.e. a di-ester of ecgonine with methanol and benzoic acid. **Reasoning:** Alkaline hydrolysis of cocaine cleaves both ester linkages to give ecgonine, methanol and benzoic acid; the liberated benzoic acid is recognised by its characteristic odour. This forms the basis of an identity test for cocaine. **Why the other options are wrong:**

- (A) Salicylic acid is not a hydrolysis product of cocaine.
- (C) Cinnamic acid arises from cinnamoylcocaine, a minor companion, not from cocaine itself.
- (D) Gallic acid is a tannin hydrolysis product, unrelated here.

**Final Answer:** Hydrolysis releases benzoic acid $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** **Go Back to Q128**

## Q129.

**Solution**

**Concept — Papaverine:** Papaverine is one of the minor opium alkaloids but, unlike morphine, it is a non-addictive smooth-muscle relaxant. **Reasoning:** Structurally papaverine is a fully aromatic 1-benzylisoquinoline (benzylisoquinoline alkaloid), which accounts for its antispasmodic and vasodilator action without narcotic activity. **Why the other options are wrong:**

- (B) Morphine, not papaverine, is the phenanthrene (morphinan) type.
- (C) Tropane alkaloids are atropine/cocaine.
- (D) Purine alkaloids are caffeine/theophylline.

**Final Answer:** Papaverine is a benzylisoquinoline alkaloid $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** **Go Back to Q129**

## Q130.

**Solution**

**Concept — Convallaria glycosides:** *Convallaria majalis* (lily of the valley) is a cardiotonic herb containing cardenolide glycosides. **Reasoning:** Its chief cardioactive glycoside is convallatoxin, a cardenolide whose aglycone strophanthidin carries the five-membered butenolide ring and is linked to L-rhamnose. **Why the other options are wrong:**

- (A) Sennoside A is an anthraquinone glycoside of senna.
- (B) Glycyrrhizin is a saponin of liquorice.
- (C) Sinigrin is a glucosinolate of mustard.

**Final Answer:** The glycoside is convallatoxin ⇒ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q130

## Q131.

**Solution**

**Concept — C-glycosides:** In a C-glycoside the sugar is bonded to the aglycone through a direct carbon–carbon bond, which is not cleaved by ordinary acid hydrolysis. **Reasoning:** Aloin (barbaloin) is an anthrone in which glucose is attached at C-10 by a C–C bond, so it is a C-glucoside and survives dilute-acid hydrolysis that would split an O-glycoside. **Why the other options are wrong:**

- (A) An O-glycoside is readily hydrolysed by acid, unlike aloin.
- (C) N-glycosides link sugar to nitrogen (e.g. nucleosides).
- (D) S-glycosides (glucosinolates) link sugar through sulphur.

**Final Answer:** Aloin is a C-glycoside (anthrone C-glucoside) ⇒ $\boxed{\text{B}}$

**Answer: (B)** Go Back to Q131

## Q132.

**Solution**

**Concept — Anthraquinone glycosides:** Hydroxyanthracene (anthraquinone) glycosides have a 1,8-dihydroxyanthraquinone aglycone joined to sugar and are responsible for the purgative action of senna, rhubarb, cascara and aloe. **Reasoning:** The schematic shows a 1,8-dihydroxyanthraquinone aglycone (the rhein/emodin type) O-linked to glucose, so the aglycone class depicted is the anthraquinone (hydroxyanthracene) derivatives. **Why the other options are wrong:**

- (A) Cardenolides are steroidal, not anthraquinone aglycones.
- (B) Cyanogenetic glycosides release HCN and have a different aglycone.
- (D) Saponins are triterpene/steroid glycosides, not anthraquinones.

**Final Answer:** The aglycone is an anthraquinone derivative ⇒ $\boxed{\text{C}}$

**Answer: (C)** Go Back to Q132

## Q133.

**Solution**

**Concept — Flavonoid glycosides:** Baicalin is the 7-O-glucuronide of the flavone baicalein from *Scutellaria baicalensis*. **Reasoning:** On hydrolysis baicalin gives glucuronic acid plus the aglycone baicalein, a trihydroxyflavone; the aglycone therefore belongs to the flavonoid (flavone) class of polyphenols. **Why the other options are wrong:**

- (B) Baicalein contains no basic nitrogen, so it is not an alkaloid.
- (C) It has no steroidal cardenolide nucleus.
- (D) It is a polyphenol, not a bitter terpenoid.

**Final Answer:** Baicalein is a flavone (flavonoid) aglycone $\Rightarrow \boxed{\text{A}}$

Answer: (A) **Go Back to Q133**

## Q134.

**Solution**

**Concept — Thyme oil:** The volatile oil of *Thymus vulgaris* is dominated by phenolic monoterpenoids. **Reasoning:** Its chief constituent is thymol, a phenolic monoterpene that is a positional isomer of carvacrol and gives thyme oil its antiseptic and antifungal properties. **Why the other options are wrong:**

- (B) Eugenol is the chief constituent of clove oil.
- (C) Geraniol typifies rose/geranium oils.
- (D) Citral characterises lemongrass oil.

**Final Answer:** Chief constituent of thyme oil is thymol $\Rightarrow \boxed{\text{A}}$

Answer: (A) **Go Back to Q134**

## Q135.

**Solution**

**Concept — Rosemary oil:** Rosemary oil from *Rosmarinus officinalis* contains cineole, camphor and $\alpha$-pinene. **Reasoning:** Its principal oxide constituent is 1,8-cineole (eucalyptol), a bicyclic monoterpene ether that is also the chief constituent of eucalyptus oil. **Why the other options are wrong:**

- (A) Anethole is from fennel/anise.

- (C) Menthol is from peppermint.
- (D) Safrole is from sassafras.

**Final Answer:** The oxide component is 1,8-cineole ⇒ $\boxed{\text{B}}$

**Answer: (B)** Go Back to Q135

## Q136.

### Solution

**Concept — Oleoresins:** An oleoresin is a homogeneous natural mixture of a resin dissolved in its own volatile (essential) oil. **Reasoning:** Copaiba exudes from *Copaifera* trunks as an oleoresin in which the resin acids are dissolved in the plant's own volatile oil, giving a clear viscous balsam-like liquid. **Why the other options are wrong:**

- (A) Gum–resin combinations contain gum, not the defining oleoresin solvent.
- (B) A fixed oil is not the natural co-solvent of an oleoresin.
- (C) Balsams (not oleoresins) are characterised by benzoic/cinnamic acids.

**Final Answer:** Copaiba is resin dissolved in its own volatile oil ⇒ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q136

## Q137.

### Solution

**Concept — Guar gum:** Guar gum from *Cyamopsis tetragonoloba* endosperm is a galactomannan polysaccharide. **Reasoning:** It has a $\beta$-(1→4)-linked D-mannose backbone bearing single D-galactose side units, so hydrolysis yields D-mannose and D-galactose in roughly a 2:1 ratio. **Why the other options are wrong:**

- (B) Glucose and rhamnose are not the galactomannan sugars.
- (C) Galacturonic acid/arabinose typify pectins and acacia.
- (D) Glucuronic acid/xylose are not the guar building blocks.

**Final Answer:** Guar gum yields D-mannose and D-galactose ⇒ $\boxed{\text{A}}$

**Answer: (A)** Go Back to Q137

## Q138.

**Solution**

**Concept — Acetate–mevalonate pathway:** Three acetyl-CoA units condense to HMG-CoA, which is reduced to mevalonic acid and then decarboxylated to the C5 isoprene units IPP and DMAPP. **Reasoning:** The scheme acetyl-CoA → HMG-CoA → mevalonic acid → IPP/DMAPP → terpenoids/steroids is exactly the acetate–mevalonate pathway, the classical route to terpenoid building blocks. **Why the other options are wrong:**

- (A) Shikimate gives aromatic amino acids, not isoprene units.
- (C) Acetate–malonate gives fatty acids and polyketide phenols.
- (D) Pentose phosphate pathway supplies sugars and NADPH.

**Final Answer:** The scheme is the acetate–mevalonate pathway $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** **Go Back to Q138**

## Q139.

**Solution**

**Concept — Shikimic acid pathway:** Erythrose-4-phosphate and phosphoenolpyruvate condense and proceed through shikimate and chorismate to the aromatic amino acids phenylalanine, tyrosine and tryptophan. **Reasoning:** From phenylalanine and tyrosine arise the C6–C3 phenylpropanoid units that build coumarins, lignans and flavonoids, so the principal products of this pathway are aromatic amino acids and phenylpropanoids. **Why the other options are wrong:**

- (A) Monoterpenes come from the mevalonate/MEP routes.
- (B) Fatty acids come from the acetate–malonate route.
- (D) Reducing sugars are products of carbohydrate metabolism.

**Final Answer:** The pathway yields phenylpropanoids and aromatic amino acids $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** **Go Back to Q139**

## Q140.

### Solution

**Concept — Sulphated ash:** The sulphated (or sulphate) ash is obtained by charring the drug, treating the carbonised residue with dilute sulphuric acid, and igniting to constant weight so that metals are converted to their sulphates. **Reasoning:**

The procedure described uses sulphuric acid on the charred sample and ignition to constant weight, which is precisely the method for sulphated ash. **Why the other options are wrong:**

- (A) Total ash is simple incineration without acid.
- (B) Acid-insoluble ash uses hydrochloric acid on the total ash.
- (C) Water-soluble ash is the water-dissolved fraction of total ash.

**Final Answer:** The sulphuric-acid procedure gives sulphated ash $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q140

## Q141.

### Solution

**Concept — Calcium oxalate crystal forms:** Calcium oxalate occurs as prisms, rosette (cluster) aggregates, raphide bundles and crystal sand, each a diagnostic powder character. **Reasoning:** A single, well-defined monoclinic crystal scattered

in the parenchyma, as sketched, is a prism crystal, typical of cinnamon bark, cinchona bark and hyoscyamus. **Why the other options are wrong:**

- (A) A rosette is a star-shaped radiating cluster, not a single prism.
- (B) Raphides are needle-shaped crystals in bundles.
- (D) Crystal sand is a mass of very small crystals (e.g. in belladonna).

**Final Answer:** The single monoclinic crystal is a prism crystal $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q141

## Q142.

### Solution

**Concept — Retardation factor ($R_f$):** $R_f$ is the ratio of the distance travelled by the solute spot to the distance travelled by the solvent front, both measured from the baseline. **Reasoning:** Here $R_f = d/D = 2.4/4.0 = 0.60$, a dimensionless value lying between 0 and 1 as required. **Why the other options are wrong:**

- (B) 1.67 is $D/d$ (the inverse) and exceeds 1, which is impossible.
- (C) 0.24 misplaces the decimal point.
- (D) 0.40 corresponds to a different migration distance.

**Final Answer:** $R_f = 2.4/4.0 = 0.60 \Rightarrow \boxed{\text{A}}$

**Answer: (A)** **Go Back to Q142**

## Q143.

**Solution**

**Concept — Overhang type of restriction enzymes:** A staggered cut can leave the protruding strand at either the 5′ end or the 3′ end; a central symmetric cut leaves blunt ends. **Reasoning:** *Pst*I recognises CTGCAG and cleaves between A and G on each strand (CTGCA↓G), so the protruding single strands are at the 3′ ends, giving 3′ overhangs. This distinguishes it from the common 5′-overhang cutters. **Why the other options are wrong:**

- (B) *Bam*HI cuts G↓GATCC, a 5′ overhang.
- (C) *Sma*I cuts CCC↓GGG centrally, blunt ends.
- (D) *Hpa*I cuts GTT↓AAC centrally, blunt ends.

**Final Answer:** *Pst*I leaves 3′ overhangs $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** **Go Back to Q143**

## Q144.

**Solution**

**Concept — Vector capacity:** Different vectors accept different insert sizes: plasmids a few kb, cosmids up to ∼45 kb, BACs up to ∼300 kb, and YACs even larger. **Reasoning:** A bacterial artificial chromosome is built on the single-copy F-factor (fertility) replicon, which keeps very large inserts (around 100–300 kb) stable with few rearrangements. For a 150 kb insert the BAC is the correct choice. **Why the other options are wrong:**

- (A) A phagemid carries only small inserts and is used for single-stranded DNA.
- (B) pUC19 is a high-copy plasmid limited to a few kb.

- (D) A $\lambda$ insertion vector accepts only a few kb of insert.

**Final Answer:** The BAC carries ∼150 kb inserts $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** **Go Back to Q144**

## Q145.

### Solution

**Concept — RT-PCR:** To clone an intron-free coding sequence, mRNA is reverse-transcribed into cDNA, which is then amplified by PCR. **Reasoning:** The first-strand synthesis from an RNA template is carried out by reverse transcriptase, an RNA-dependent DNA polymerase (originally from retroviruses such as AMV or MMLV). The resulting cDNA lacks introns, so the eukaryotic protein can be expressed in a bacterial host. **Why the other options are wrong:**

- (A) *Taq* polymerase copies DNA, not RNA, and is used in the later PCR cycling.
- (C) Terminal transferase adds nucleotides to $3'$ ends without a template.
- (D) RNA polymerase II makes RNA from DNA, the reverse direction.

**Final Answer:** Reverse transcriptase makes the first cDNA strand $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** **Go Back to Q145**

## Q146.

### Solution

**Concept — Recombinant interferon-$\beta$:** Type I interferons are recombinant cytokines; interferon-$\beta$ is licensed as a disease-modifying therapy for multiple sclerosis. **Reasoning:** Recombinant interferon beta-1a (mammalian cell) and beta-1b (*E. coli*) reduce relapse frequency in relapsing–remitting multiple sclerosis by immunomodulation. None of the other listed proteins is used for MS. **Why the other options are wrong:**

- (B) Recombinant insulin treats diabetes mellitus.
- (C) Recombinant factor VIII treats haemophilia A.
- (D) Erythropoietin treats anaemia of renal failure.

**Final Answer:** Interferon-$\beta$ is the MS disease-modifier $\Rightarrow \boxed{\text{A}}$

**Answer: (A)** **Go Back to Q146**

## Q147.

### Solution

**Concept — Selectable marker:** An antibiotic-resistance gene on a vector lets only successfully transformed cells survive on antibiotic-containing medium, separating them from untransformed cells. **Reasoning:** The $kan^R$ gene encodes an enzyme that inactivates kanamycin. Only cells carrying the plasmid (and therefore $kan^R$) form colonies on kanamycin plates; untransformed cells die. Its job is selection, distinct from the *ori*, promoter, and MCS shown on the map. **Why the other options are wrong:**

- (A) The origin (*ori*), not $kan^R$, starts replication.
- (B) The promoter P drives transcription of the insert.
- (C) The MCS is the insertion site for foreign DNA.

**Final Answer:** $kan^R$ lets only transformants grow on kanamycin $\Rightarrow \boxed{\text{D}}$

**Answer: (D)** **Go Back to Q147**

## Q148.

### Solution

**Concept — Lactic acid fermentation:** Homofermentative lactic acid bacteria convert sugars almost entirely to lactic acid, the basis of its industrial production. **Reasoning:** *Lactobacillus delbrueckii* (and related species) ferment glucose, whey lactose, or molasses to give high yields of lactic acid, which is used for polylactic-acid plastics, food acidulants, and pharmaceuticals. It is the classic industrial producer. **Why the other options are wrong:**

- (A) *Aspergillus niger* makes citric acid, not lactic acid.
- (C) *Saccharomyces cerevisiae* produces ethanol.
- (D) *Acetobacter aceti* oxidises ethanol to acetic acid (vinegar).

**Final Answer:** *Lactobacillus delbrueckii* produces lactic acid $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** **Go Back to Q148**

## Q149.

### Solution

**Concept — Riboflavin (vitamin B$_2$) fermentation:** Certain flavinogenic fungi overproduce riboflavin and are used for its commercial manufacture. **Reasoning:**

*Ashbya gossypii* is a natural riboflavin overproducer and is the principal industrial organism for vitamin B$_2$, displacing chemical synthesis. (*Eremothecium ashbyi* is a related producer.) **Why the other options are wrong:**

- (A) *Penicillium chrysogenum* produces penicillin.
- (B) *Streptomyces griseus* produces streptomycin.
- (D) *Clostridium acetobutylicum* carries out the acetone–butanol fermentation.

**Final Answer:** *Ashbya gossypii* overproduces riboflavin $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q149

## Q150.

### Solution

**Concept — Dextran production:** Dextran is a branched $\alpha$-1,6 glucan synthesised extracellularly from sucrose by dextransucrase. **Reasoning:** *Leuconostoc mesenteroides* secretes dextransucrase, which polymerises the glucose moiety of sucrose into dextran (releasing fructose). The product, after controlled hydrolysis to a defined molecular weight, serves as a plasma volume expander. **Why the other options are wrong:**

- (B) *E. coli* is not the industrial dextran producer.
- (C) *Bacillus subtilis* is used for enzymes and levan/$\gamma$-PGA, not dextran.
- (D) *Candida utilis* is grown for single-cell protein.

**Final Answer:** *Leuconostoc mesenteroides* makes dextran $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q150

## Q151.

### Solution

**Concept — $K_m$ and affinity:** $K_m$ is the [S] giving half-maximal velocity; a *lower* $K_m$ means the enzyme reaches half $V_{max}$ at low substrate, i.e. higher affinity for the substrate. **Reasoning:** $E_1$ reaches $\frac{1}{2}V_{max}$ at a smaller [S] than $E_2$, so $K_m(E_1) < K_m(E_2)$. The smaller $K_m$ corresponds to the tighter binding, so $E_1$ has the higher affinity for the substrate. Equal $V_{max}$ values do not affect this comparison. **Why the other options are wrong:**

- (A) A larger $K_m$ means lower, not higher, affinity.
- (C) Equal $V_{max}$ does not imply equal $K_m$ or equal affinity.
- (D) Affinity is read directly from $K_m$ on this plot.

**Final Answer:** Smaller $K_m$ of $E_1$ means higher affinity $\Rightarrow \boxed{\text{B}}$

**Answer: (B)** **Go Back to Q151**

## Q152.

### Solution

**Concept — Uncompetitive inhibition:** An uncompetitive inhibitor binds only the enzyme–substrate complex, lowering both apparent $K_m$ and $V_{max}$ by the same factor; on a Lineweaver–Burk plot this gives *parallel* lines. **Reasoning:** Because the slope of a double-reciprocal line is $K_m/V_{max}$ and both fall by the same factor, the slope is unchanged, so the inhibited and uninhibited lines are parallel (different intercepts, same slope). This is the signature of uncompetitive inhibition. **Why the other options are wrong:**

- (A) Inhibitors do not raise $V_{max}$.
- (B) A competitive inhibitor raises $K_m$ with $V_{max}$ unchanged and gives intersecting lines on the $1/v$ axis.
- (D) Non-competitive inhibition lowers $V_{max}$ with $K_m$ unchanged, giving lines that intersect on the $1/[S]$ axis.

**Final Answer:** Parallel lines mean $K_m$ and $V_{max}$ both fall proportionally (uncompetitive) $\Rightarrow \boxed{\text{C}}$

**Answer: (C)** **Go Back to Q152**

## Q153.

### Solution

**Concept — Secretory IgA:** IgA is the dominant antibody of external secretions, where it occurs as a dimer linked by a J chain and carrying a secretory component that protects it from proteolysis. **Reasoning:** Secretory IgA coats mucosal surfaces (gut, respiratory tract), saliva, tears, and colostrum, neutralising pathogens and toxins at the point of entry. The secretory component is acquired during transcytosis across epithelial cells. **Why the other options are wrong:**

- (A) IgG dominates blood and tissue, not secretions.
- (B) IgM is the pentameric first-response antibody of plasma.
- (C) IgE mediates allergy and is present only in traces.

**Final Answer:** The mucosal secretory antibody is IgA $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q153

## Q154.

### Solution

**Concept — Monoclonal antibody nomenclature:** The sub-stem before `-mab` encodes the source: `-omab` murine, `-ximab` chimeric, `-zumab` humanised, `-umab` fully human. **Reasoning:** A fully human monoclonal antibody such as adalimumab carries the sub-stem `-umab`. Recognising the increasing “humanness” from murine to human across these sub-stems is the key to the question. **Why the other options are wrong:**

- (A) `-omab` denotes a fully murine antibody (e.g. ibritumomab).
- (B) `-ximab` denotes a chimeric antibody (e.g. rituximab).
- (D) `-zumab` denotes a humanised antibody (e.g. trastuzumab).

**Final Answer:** Fully human antibodies use `-umab` $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q154

## Q155.

### Solution

**Concept — Alternative complement pathway:** The alternative pathway is an antibody-independent, innate route triggered directly on microbial surfaces by spontaneous “tickover” hydrolysis of C3, and stabilised by factors B, D, and properdin. **Reasoning:** Unlike the classical pathway (which needs antigen–antibody complexes and C1q) and the lectin pathway (which needs mannose-binding lectin), the alternative pathway starts when low-level C3 hydrolysis deposits C3b on a microbe; factor B, factor D, and properdin then form the C3 convertase. All three pathways converge on C3 and build the membrane-attack complex. **Why the other options are wrong:**

- (A) Antibody–C1q activation is the classical pathway.
- (C) Mannose-binding lectin defines the lectin pathway.
- (D) The alternative pathway does form the membrane-attack complex.

**Final Answer:** It is antibody-independent, surface-triggered via factors B, D, properdin $\Rightarrow \boxed{B}$

**Answer: (B)** Go Back to Q155

## Q156.

### Solution

**Concept — Type III hypersensitivity:** Type III reactions are caused by soluble immune complexes that deposit in tissues, fix complement, and recruit neutrophils, producing inflammation and damage. **Reasoning:** Serum sickness and the localised Arthus reaction are the textbook examples of immune-complex (Type III) hypersensitivity. The deposited antigen–antibody complexes activate complement (generating C3a/C5a), attracting neutrophils whose enzymes injure vessel walls and joints. **Why the other options are wrong:**

- (A) Type I is immediate, IgE-mediated (e.g. anaphylaxis).
- (B) Type II is antibody-mediated cytotoxicity against cell-surface antigens.
- (C) Type IV is delayed, T-cell-mediated (e.g. tuberculin reaction).

**Final Answer:** Immune-complex serum sickness/Arthus is Type III $\Rightarrow \boxed{D}$

**Answer: (D)** Go Back to Q156

## Q157.

### Solution

**Concept — Capsule (negative) staining:** The capsule is a gelatinous layer that does not take up ordinary stains, so it is shown by staining the background and the cell while the capsule stays clear. **Reasoning:** In India-ink or Anthony's method the background is darkened and the cell body coloured, leaving the capsule as a bright, unstained halo around each cell. The capsule resists the dye because of its hydrated polysaccharide nature. **Why the other options are wrong:**

- (B) A stained terminal body describes an endospore stain, not a capsule.
- (C) Radiating stained filaments describe a flagella stain.
- (D) Intracellular stained granules are metachromatic/PHB inclusions.

**Final Answer:** The capsule is a clear halo on a dark background $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q157

## Q158.

### Solution

**Concept — z-value:** The z-value links heat resistance to temperature: it is the temperature rise needed to cut the decimal-reduction time (D-value) to one-tenth. **Reasoning:** While D measures killing rate at a fixed temperature, z (in °C) measures how strongly D depends on temperature. A typical z of about 10°C means raising the temperature by 10°C reduces D tenfold, the basis for calculating $F_0$ in steam sterilisation. **Why the other options are wrong:**

- (A) Reducing the count by one log cycle at fixed temperature is the D-value.
- (C) The starting number of organisms is the bioburden.
- (D) Equivalent minutes at 121°C is the $F_0$ value.

**Final Answer:** z is the °C change that alters D tenfold $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q158

## Q159.

### Solution

**Concept — Depyrogenation:** Endotoxins are heat-stable lipopolysaccharides that survive autoclaving; their destruction (depyrogenation) requires high dry heat. **Reasoning:** Glassware is depyrogenated by dry heat at about 250°C for 30 min (or a validated cycle giving at least a 3-log reduction in endotoxin). This both sterilises and chemically degrades the pyrogen, which ordinary 121°C steam cannot do. **Why the other options are wrong:**

- (A) Autoclaving at 121°C sterilises but does not destroy endotoxin.
- (B) Filtration removes microbes but not dissolved endotoxin from glass.
- (D) Ethylene oxide is a sterilant for plastics, not a depyrogenation method.

**Final Answer:** Dry heat at 250°C for 30 min depyrogenates glassware $\Rightarrow \boxed{\text{C}}$

Answer: (C) Go Back to Q159

## Q160.

### Solution

**Concept — LAL test:** The horseshoe-crab (*Limulus*) amoebocyte lysate contains a clotting-enzyme cascade that is activated specifically by bacterial endotoxin, producing a gel clot. **Reasoning:** Endotoxin activates Factor C of the lysate, triggering a protease cascade that converts coagulogen to coagulin and forms a gel (gel-clot method) or a measurable turbidity/colour (kinetic methods). This sensitive, specific reaction detects endotoxin in injectables and water for injection. **Why the other options are wrong:**

- (A) The test does not lyse bacteria; it responds to endotoxin even from dead cells.
- (B) It is specific for endotoxin, not total protein.
- (C) $\beta$-glucan reactivity is a known interference, but the LAL test is used for endotoxin.

**Final Answer:** Endotoxin triggers a clotting cascade that gels the lysate $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q160

## Answer Key

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