GUJCET Biology

Sample Paper – 10

Duration: 60 Minutes **Maximum Marks: 40**

Instructions

- This paper contains **40** Multiple Choice Questions (Single Correct Answer), modelled on the Biology portion of **GUJCET** (Gujarat Common Entrance Test).
- Each correct answer carries **+1 mark**. There is a negative marking of -0.25 **marks** for each incorrect answer. Unanswered questions carry no penalty.
- Only **one** option is correct per question. Mark responses on the OMR sheet carefully.
- Syllabus level: **GSEB Class 11–12 Biology (NCERT aligned) — Human Physiology, Plant Physiology, Reproduction, Genetics, Ecology, Biotechnology.**
- Use of mobile phones, calculators, or any electronic device is strictly prohibited.

Q1. The disaccharide sucrose (table sugar) is digested in the small intestine by which enzyme, and where exactly is this enzyme found?

A. Sucrase (sucrase-isomaltase), located on the brush border membrane of small intestinal enterocytes

B. Salivary amylase, secreted by the salivary glands and active in the oral cavity

C. Pancreatic amylase, released into the duodenal lumen from the exocrine pancreas

D. Sucrase secreted by the exocrine pancreas directly into the intestinal lumen

Q2. Emulsification of dietary fats by bile salts is important for lipid digestion because:

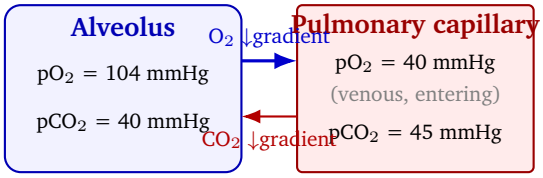
A. Bile salts chemically hydrolyse triglycerides into fatty acids and glycerol

B. Emulsification breaks large fat globules into smaller micelle-sized droplets, greatly increasing the surface area available for pancreatic lipase to act upon

C. Bile salts directly activate the enzyme pancreatic lipase by changing its tertiary conformation

D. Emulsification converts triglycerides into phospholipids, which are then absorbed directly

Q3. Study the diagram below showing partial pressures of respiratory gases in the alveolus and pulmonary capillary.



According to Dalton's law of partial pressures, O_2 diffuses from the alveolus into the pulmonary capillary blood because:

A. The respiratory pigment haemoglobin actively pumps O_2 across the alveolar membrane using ATP

B. The partial pressure of CO_2 in alveolar air is higher than in venous blood, driving O_2 exchange

C. The partial pressure of O_2 in alveolar air ($\approx$104 mmHg) is higher than in venous blood ($\approx$40 mmHg), so O_2 moves down its pressure gradient from alveolus to blood

D. The total barometric pressure at the alveolus is higher than at the pulmonary capillary

Q4. A person diagnosed with pulmonary fibrosis (a restrictive lung disease in

which lung tissue becomes stiff and scarred) would be expected to show which of the following changes in lung volumes?

A. An isolated increase in residual volume (RV) due to gas trapping in fibrotic zones

B. A selective decrease in functional residual capacity (FRC) only, with all other volumes unchanged

C. An increase in total lung capacity (TLC) only, as fibrosis traps air in non-compliant segments

D. A decrease in both vital capacity (VC) and total lung capacity (TLC), because fibrosis reduces lung compliance and restricts full expansion of the lungs

Q5. Von Willebrand factor (vWF), a large plasma glycoprotein, plays a critical role in primary haemostasis. Which statement CORRECTLY describes its function?

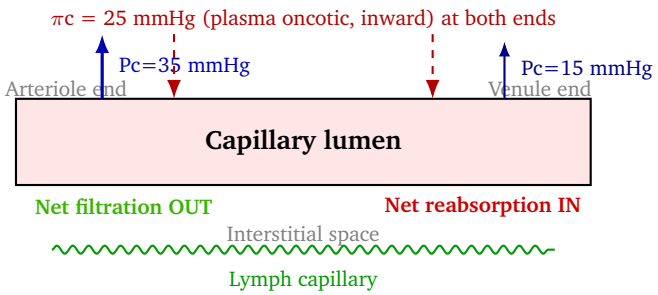
A. It bridges exposed subendothelial collagen to the platelet surface receptor GPIb/IX, thereby facilitating platelet adhesion to the damaged vessel wall

B. It directly activates coagulation factor X (Stuart–Prower factor) in the common coagulation pathway

C. It is the precursor of fibrin and is cleaved by thrombin to form the permanent haemostatic plug

D. It stimulates the liver to release thrombopoietin, which promotes platelet production in the bone marrow

Q6. The diagram below illustrates Starling forces acting across a capillary wall.



Oedema (tissue swelling) develops when:

A. Capillary hydrostatic pressure decreases well below the plasma oncotic pressure, so fluid is drawn into the capillary

B. Plasma oncotic pressure decreases (e.g. due to hypoalbuminaemia) or capillary hydrostatic pressure increases, causing net fluid accumulation in the interstitium

C. Increased lymphatic flow accelerates removal of excess fluid from interstitial tissues

D. Arterial blood pressure falls sharply, reducing capillary filtration and dehydrating tissues

Q7. The baroreceptor reflex (baroreflex) is a rapid autonomic mechanism for maintaining arterial blood pressure. Which statement CORRECTLY explains how it works?

A. Baroreceptors located in the left ventricular wall directly sense stroke volume and adjust cardiac output accordingly

B. When blood pressure falls, baroreceptors stimulate the adrenal medulla to release large amounts of epinephrine as the primary regulatory mechanism

C. Baroreceptors in the carotid sinus (via CN IX) and aortic arch (via CN X) detect changes in arterial stretch and signal the cardiovascular control centre in the medulla oblongata, which adjusts heart rate and peripheral vascular resistance

D. Baroreceptors directly trigger renin release from the juxtaglomerular apparatus of the kidney as the sole mechanism to restore blood pressure

Q8. A 45-year-old male presents to a urology clinic with severe flank pain radiating to the groin, haematuria, and crystalluria. Imaging reveals a radio-opaque stone in the right ureter. Which type of kidney stone (nephrolithiasis) is MOST frequently diagnosed in clinical practice?

A. Uric acid crystals, found predominantly in patients with gout or high purine diets

B. Struvite (magnesium ammonium phosphate) stones, caused exclusively by urease-producing bacteria

C. Cystine crystals, resulting from a rare autosomal recessive defect in renal amino acid transport

D. Calcium oxalate stones, the most frequently encountered type, associated with hypercalciuria or hyperoxaluria

Q9. A marine teleost fish (a bony fish such as a herring) lives in seawater, which is more concentrated (hypertonic) than its body fluids. How does it maintain water and salt balance?

A. It drinks seawater continuously, actively excretes excess NaCl through specialised gill ionocytes (chloride cells), and produces small volumes of concentrated urine

B. It drinks seawater in very small amounts to slightly raise internal osmolarity, relying mainly on skin impermeability for water retention

C. It produces large volumes of dilute urine to flush out excess salts absorbed passively from seawater

D. It absorbs ions from the surrounding seawater passively through the skin by simple diffusion to equilibrate with the environment

Q10. The loop of Henle in the mammalian nephron is essential for producing concentrated urine. What is the primary function of the counter-current multiplier system in this structure?

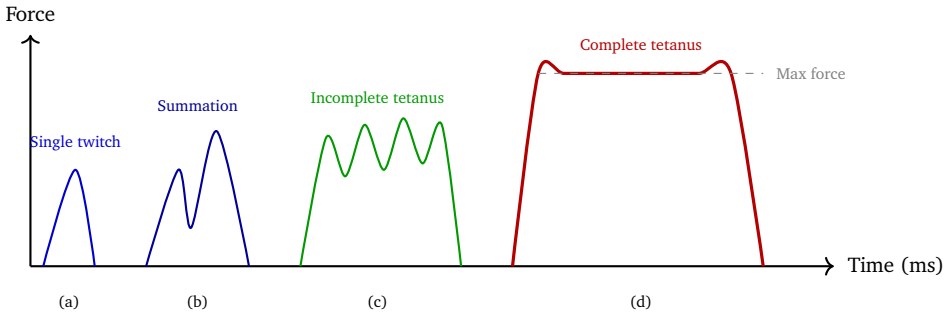
A. To dilute the filtrate by adding water to it in the thin ascending limb, producing a hypotonic filtrate before it reaches the distal tubule

B. To generate a hyperosmotic medullary interstitium that enables the collecting duct (under ADH influence) to reabsorb water and concentrate the final urine

C. To reabsorb glucose and amino acids from the filtrate that escaped reabsorption in the proximal convoluted tubule

D. To secrete H^+ and K^+ ions into the filtrate to regulate blood pH and plasma potassium concentration

Q11. The graph below shows muscle force responses to stimuli delivered at increasing frequencies.



Complete tetanic contraction in skeletal muscle (as shown in panel d) occurs when:

A. Each individual twitch has completely relaxed before the next stimulus arrives, producing maximal force per stimulus

B. The muscle has become fatigued, and lactic acid accumulation has locked the cross-bridges in the contracted position

C. Stimuli arrive at such a high frequency that successive twitches fuse into a sustained, smooth contraction with no relaxation between them

D. Calcium is completely and irreversibly depleted from the sarcoplasmic reticulum, maintaining troponin in its active conformation

Q12. A sports physiologist compares two athletes: a marathon runner (42 km) and a 100 m sprinter. Which statement CORRECTLY describes the predominant muscle fibre type used by the marathon runner, and the physiological reason for this?

A. Fast-twitch (Type IIb) fibres, because they generate the highest peak force and power output per contraction

B. Fast-twitch (Type IIa) fibres, because they employ both oxidative and glycolytic metabolism for intermediate endurance

C. Slow-twitch (Type I) fibres are not suitable; the marathon runner uses glycolytic fibres to sustain pace over the full distance

D. Slow-twitch (Type I) fibres, because they are rich in mitochondria, rely predominantly on oxidative phosphorylation, and are highly resistant to fatigue during prolonged aerobic activity

Q13. A 52-year-old patient presents with a wide-based, staggering gait (ataxia), inability to perform rapid alternating hand movements (dysdiadochokinesia), and overshooting when reaching for objects (dysmetria, or past-pointing). MRI reveals a lesion in which brain region?

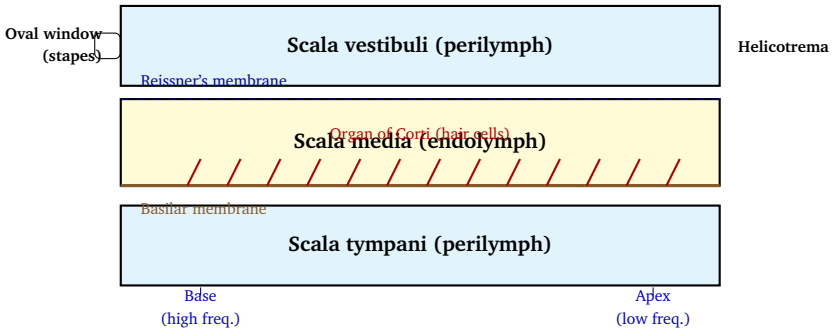
A. Cerebellum

B. Primary motor cortex (precentral gyrus), which initiates voluntary motor commands

C. Basal ganglia, which suppress unwanted movements and regulate movement initiation

D. Thalamus, the primary sensory relay nucleus connecting the spinal cord to the cortex

Q14. The diagram below shows a cross-section of the unrolled cochlea.



Sound frequencies are detected at different positions along the basilar membrane in the cochlea because:

A. Hair cells at the apex are activated by high-frequency sounds while hair cells at the base respond to low-frequency sounds

B. The basilar membrane varies in width and stiffness along its length — narrow and stiff at the base (resonates with high frequencies) and wide and flexible at the apex (resonates with low frequencies)

C. The tympanic membrane vibrates at different amplitudes for different frequencies, selectively activating distinct zones of the cochlea

D. The ossicles (malleus, incus, stapes) selectively transmit only one frequency at a time based on the footplate area of the stapes

Q15. The anterior pituitary releases gonadotrophins FSH and LH that regulate gonadal function. In males, which statement CORRECTLY pairs each hormone with its specific testicular target cell and function?

A. FSH targets Leydig (interstitial) cells to stimulate testosterone synthesis and secretion

B. LH (ICSH) targets Sertoli (sustentacular) cells within the seminiferous tubules to nurture developing sperm

C. FSH acts on Sertoli cells to support spermatogenesis (by producing androgen-binding protein and inhibin); LH (ICSH) acts on Leydig cells to stimulate testosterone production

D. Both FSH and LH act on Leydig cells, but with different second messenger cascades producing distinct downstream effects

Q16. Oxytocin is released from the posterior pituitary during parturition and creates a positive feedback loop. Which statement CORRECTLY explains the Ferguson reflex?

A. Oxytocin directly stimulates oestrogen synthesis in the corpus luteum, which further amplifies oxytocin secretion from the anterior pituitary

B. Oxytocin suppresses progesterone secretion, removing progesterone's inhibitory effect on uterine contractions and creating positive feedback

C. Oxytocin is secreted at a constant, continuous rate throughout pregnancy, gradually building up to trigger contractions at term

D. Uterine contractions caused by oxytocin stretch the cervix further, which activates mechanoreceptors that signal the hypothalamus to release more oxytocin — this continues as a positive feedback loop until the baby is delivered

Q17. During receptor-mediated endocytosis (e.g., the LDL receptor pathway in hepatocytes), what is the fate of the receptor protein after internalisation?

A. The receptor dissociates from the LDL in the acidic environment of the early endosome and is recycled back to the plasma membrane in recycling vesicles

B. The receptor is degraded together with the LDL cholesterol particle inside the lysosome, requiring the cell to synthesise fresh receptor molecules

C. The receptor is transported retrogradely to the Golgi apparatus for permanent storage and glycosylation before re-use

D. The receptor is exported from the cell via exocytosis after releasing the LDL into the cytoplasm

Q18. A pathologist examining a tissue sample notes cells that have shrunk, with densely condensed chromatin and intact plasma membranes, and the cells have been phagocytosed cleanly without surrounding inflammation. Which feature CORRECTLY distinguishes this mode of cell death from necrosis?

A. This process involves cell swelling, membrane rupture, and release of intracellular contents that trigger a strong inflammatory response — a hallmark of necrosis, not what is observed

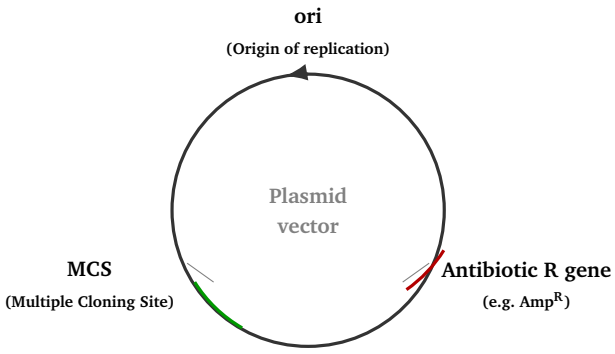
B. Apoptosis results in the neat fragmentation of the cell into membrane-bound apoptotic bodies that are phagocytosed without triggering inflammation, and is mediated by caspase proteases (caspases 3, 6, 7

as executioners)

C. Necrosis is an energy-dependent, programmed process that requires caspase activation and produces apoptotic bodies

D. Apoptosis can only be triggered by external immune-cell signals (extrinsic pathway) and cannot be initiated by internal damage such as DNA double-strand breaks

Q19. A molecular biologist wishes to clone a human gene into a bacterial host. The diagram below shows a plasmid cloning vector.



A plasmid used as a cloning vector in recombinant DNA technology must possess:

A. A centromere, telomeres, and associated histone proteins for stable autonomous replication in bacteria

B. Only an antibiotic resistance gene is necessary, since plasmids are maintained solely by antibiotic selection pressure

C. An origin of replication (ori) for autonomous replication, a selectable marker (e.g., antibiotic resistance gene) for identifying transformed cells, and a multiple cloning site (MCS) for insertion of foreign DNA

D. A nuclear membrane-like structure to protect the recombinant DNA from cytoplasmic nucleases in the bacterial host

Q20. A small group of 30 individuals from a large mainland bird population colonises a remote offshore island. Several alleles present in the mainland population are absent in these 30 founders by chance. This illustrates which evolutionary mechanism?

A. The bottleneck effect, where a drastic reduction in a pre-existing population randomly preserves only a subset of alleles

B. Natural selection acting on a small, isolated population to fix beneficial mutations more rapidly than in a large population

C. Gene flow, where migrants carry new alleles into the island population, increasing genetic diversity

D. The founder effect — a small, non-representative subset of the original gene pool establishes a new population with reduced allele diversity and allele frequencies that differ from the source population

Q21. Bat wings, bird wings, and insect wings each enable flight, yet they are constructed from fundamentally different materials and embryonic tissues. These structures are therefore classified as analogous. Which explanation is CORRECT?

A. They have similar function (powered flight) but evolved independently from different ancestral structures in unrelated lineages, exemplifying convergent evolution

B. They share a common ancestral developmental pathway, with the same embryonic forelimb bones modified differently in each lineage

C. They have essentially the same internal bone arrangement (radius, ulna, humerus), confirming descent from a single winged ancestor

D. All three evolved from the same ancestral wing structure present in early Carboniferous tetrapods before the amniote radiation

Q22. In a monohybrid cross experiment, 100 F_2 offspring are scored: 80 show the dominant phenotype and 20 show the recessive phenotype. The expected 3:1 Mendelian ratio predicts 75 dominant and 25 recessive. The chi-square value is $\chi^2 = \frac{(80-75)^2}{75} + \frac{(20-25)^2}{25} = 0.33 + 1.00 = 1.33$. At 1 degree of freedom the critical value is 3.84 ($p = 0.05$). The conclusion is:

A. Statistically significant — the observed data deviate significantly from

the expected 3:1 ratio, so a different inheritance pattern must be operating

B. Not statistically significant — the deviation ($1.33 < 3.84$) falls within expected random variation, and the 3:1 Mendelian ratio cannot be rejected

C. Statistically significant simply because the observed dominant count (80) exceeds the expected count (75)

D. Inconclusive — at least 500 offspring must be scored before the chi-square test can be applied to genetic data

Q23. A plant geneticist performs two reciprocal crosses: (i) Female plant with variegated leaves $\times$ Male plant with green leaves; (ii) Female plant with green leaves $\times$ Male plant with variegated leaves. In cross (i) all offspring are variegated; in cross (ii) all offspring are green. This pattern indicates:

A. Nuclear gene inheritance following Mendel's laws, because the result is the same regardless of which parent supplies which allele

B. Paternal (cytoplasmic) inheritance — offspring always adopt the father's phenotype, regardless of the mother's genotype

C. Cytoplasmic (maternal) inheritance — reciprocal crosses give different results because the offspring's phenotype is determined by the maternal parent's cytoplasm (in this case, plastid genotype), not the paternal nuclear genome

D. Sex-linked inheritance — the trait is carried on the X chromosome and shows different ratios in reciprocal crosses only in male offspring

Q24. Telomerase is a ribonucleoprotein enzyme (reverse transcriptase) that extends telomeric DNA at chromosome ends. Why is telomerase considered a promising target for anticancer therapy?

A. Telomerase synthesises new DNA during normal S-phase replication at origins of replication to proofread and repair mismatches

B. Telomerase directly ligates double-strand breaks at non-telomeric sites, promoting genomic stability in dividing cells

C. Because all somatic cells actively express telomerase, inhibiting it would selectively kill cancer cells without any side-effects on normal tissues

D. Most cancer cells overexpress telomerase to maintain telomere length, enabling replicative immortality (a hallmark of cancer); inhibiting telomerase causes progressive telomere shortening, triggering senescence or apoptosis specifically in cells that divide indefinitely

Q25. Post-translational modifications (PTMs) alter the activity, localisation, or stability of proteins after their synthesis on the ribosome. Which of the following is a CORRECT example of a PTM and its functional consequence?

A. Phosphorylation of serine, threonine, or tyrosine residues by protein kinases, which can activate or inactivate enzymes and is a central mechanism of intracellular signal transduction

B. Translation of mRNA by the ribosome, producing a primary polypeptide chain from its amino acid sequence — this is itself the modification

C. Removal of introns from pre-mRNA by the spliceosome before the mRNA is exported from the nucleus for translation

D. Methylation of the 5′ 7-methylguanosine cap on mRNA to protect the transcript from exonucleolytic degradation in the cytoplasm

Q26. Histone acetylation is an important epigenetic modification that regulates gene expression. Which explanation CORRECTLY describes why histone acetylation promotes transcription?

A. Acetylation physically removes the histone octamer from the nucleosome core, leaving the DNA completely free of protein

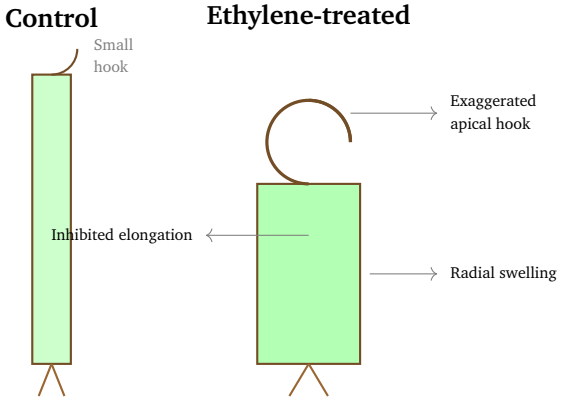
B. Acetylation of lysine residues on histone tails neutralises their positive charge, weakening histone–DNA electrostatic interactions and

creating open (euchromatic) chromatin that is accessible to transcription factors and RNA polymerase II

C. Acetylation adds large, bulky carbohydrate groups to histones, physically pushing DNA strands away from the histone core

D. Histone acetyltransferases (HATs) simultaneously methylate the adjacent gene promoter CpG island, directly marking it for active transcription

Q27. Ethylene is a gaseous plant hormone. The diagram below compares an untreated seedling with an ethylene-treated etiolated seedling.



The triple response of etiolated dicot seedlings to ethylene is characterised by:

A. Rapid hypocotyl elongation, increased primary root growth, and straightening of the apical hook to expose the apical meristem to light

B. Expansion of the leaf lamina, greening of the cotyledons due to chloroplast development, and induction of flowering

C. Inhibition of hypocotyl elongation, radial swelling of the hypocotyl (making it shorter and thicker), and exaggerated apical hook formation

D. Rapid fruit ripening, leaf abscission at the petiole base, and phototropic bending of the stem toward the light source

Q28. Plant roots actively absorb mineral ions (such as K^+, NO_3^-, $H_2PO_4^-$) against their electrochemical gradients from the soil solution. What is the primary driving force for this active ion uptake in root hair cells?

A. The Na^+/K^+-ATPase pump, which establishes a transmembrane Na^+ gradient used to drive ion uptake as in animal cells

B. Osmosis, because water flowing into the root hair cells by osmotic pressure carries dissolved mineral ions along with it

C. ATP molecules are physically attached to ion transport proteins, which carry individual ions across the membrane using the chemical energy stored in ATP directly

D. The proton motive force generated by the plasma membrane H^+-ATPase, which pumps H^+ out of the cell, creating a negative membrane potential (inside negative) and a H^+ gradient that drives cation (K^+) uptake via specific channels and H^+-cotransporters (symporters)

Q29. During germination of oil-rich seeds (e.g., castor bean, sunflower), stored triacylglycerols are converted to sucrose for use by the growing seedling. Which statement CORRECTLY identifies the organelles involved and describes the metabolic pathway?

A. Glyoxysomes (specialised peroxisomes) carry out beta-oxidation of fatty acids and the glyoxylate cycle, converting acetyl-CoA to succinate, which is exported to mitochondria and used for gluconeogenesis to produce sucrose

B. Chloroplasts and vacuoles collaborate, using light energy absorbed by chlorophyll to directly re-synthesise glucose from fatty acid chains

C. The nucleus and rough ER contain enzymes that transcribe fatty acid sequences into glucose polymers, which are then packaged by the Golgi

D. The Golgi apparatus and smooth ER reverse the esterification of fatty acids from glycerol to regenerate free glucose molecules for transport to the embryo axis

Q30. The Calvin cycle (light-independent reactions) occurs in the stroma of the chloroplast and fixes atmospheric CO_2 into organic molecules. Which

enzyme catalyses the initial carboxylation step, and what is the immediate product?

A. Phosphoglycerate kinase (PGK) adds CO_2 to ATP, producing 3-phosphoglycerat in an energy-dependent step

B. Ribulose-1,5-bisphosphate carboxylase/oxygenase (RuBisCO), which adds CO_2 to ribulose-1,5-bisphosphate (RuBP) to yield two molecules of 3-phosphoglycerate (3-PGA) as the immediate product

C. Ferredoxin-$NADP^+$ reductase (FNR), which transfers electrons to CO_2 and reduces it to 3-PGA using NADPH from the light reactions

D. Phosphoribulokinase (PRK), which phosphorylates ribulose-5-phosphate to regenerate RuBP and simultaneously incorporates CO_2

Q31. A couple seek contraceptive advice and choose a copper-T intrauterine device (IUD). How does the copper-T IUD prevent pregnancy?

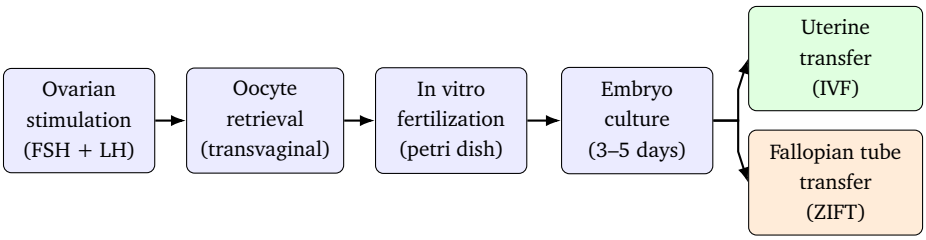
A. It acts as a physical cervical plug, preventing sperm from entering the uterine cavity

B. It releases a sustained dose of synthetic oestrogen that suppresses FSH and LH secretion from the anterior pituitary, thereby inhibiting ovulation

C. Copper ions continuously released by the device are toxic to spermatozoa (spermicidal — inhibiting motility and viability), and the device also creates an inhospitable endometrial environment that may prevent implantation of a fertilised egg

D. It raises the pH of cervical mucus to an alkaline level, which is lethal to sperm that require an acidic environment for capacitation

Q32. The diagram below shows the steps of assisted reproductive technology (ART).



In ZIFT (Zygote Intrafallopian Transfer), the embryo is transferred at which developmental stage and to which anatomical location?

A. At the blastocyst stage (approximately 5 days after fertilization) into the uterine cavity via a transcervical catheter

B. At the morula stage (16 cells) through the cervix for natural descent and implantation in the uterus

C. At the 8-cell stage via transcervical catheter into the uterine fundus, bypassing the fallopian tube

D. At the zygote stage (immediately after fertilization in vitro, before cleavage) into the fallopian tube by laparoscopy

Q33. The placenta is a haemochorial organ that develops from the trophoblast and serves multiple functions during pregnancy. Which of the following CORRECTLY describes a critical endocrine function of the placenta?

A. From approximately 10–12 weeks of gestation, the placenta takes over from the corpus luteum as the principal source of progesterone and oestrogen, thereby maintaining the uterine endometrium throughout the remainder of pregnancy

B. The placenta secretes FSH and LH to continuously stimulate the ovary, maintaining a follicular phase throughout gestation to support the developing fetus

C. The placenta produces glucagon as a counter-regulatory hormone to prevent hypoglycaemia in the mother during periods of prolonged fasting in late pregnancy

D. The placenta secretes renin and angiotensin II to exclusively regulate maternal blood pressure and prevent pre-eclampsia

Q34. Allopatric speciation and sympatric speciation are the two major modes by which new species arise. Allopatric speciation is MOST likely to occur when:

A. A single mutant individual arises within a population that is repro-

ductively isolated from all other members due to a novel chromosome rearrangement

B. A geographical barrier such as a mountain range, ocean, or river physically separates a population into two or more groups that then diverge genetically through natural selection and genetic drift, eventually becoming reproductively isolated

C. A population grows so large that resource competition drives disruptive selection, causing the population to split into ecotypes within the same habitat

D. Chromosomal polyploidy doubles the chromosome number in a subgroup of plants living within the original habitat, instantly creating reproductive isolation from diploid individuals

Q35. The ecologist G.F. Gause conducted classic competition experiments with *Paramecium aurelia* and *P. caudatum* in laboratory cultures. What principle did his results demonstrate?

A. Predation is the primary force preventing a single species from outcompeting all others and monopolising resources in any ecosystem

B. Two strongly competing species will evolve to become phenotypically more similar over time through character convergence

C. Two species that occupy the same fundamental ecological niche and compete for identical resources cannot stably coexist — one will competitively exclude the other over time (Gause's competitive exclusion principle)

D. Interspecific competition always results in the complete extinction of one species and exponential population growth of the other within one generation

Q36. Decomposition of dead organic matter (detritus) involves several sequential sub-processes. **Humification** specifically refers to:

A. The physical fragmentation of large leaf litter and dead wood into smaller pieces by detritivores such as earthworms, millipedes, and

woodlice

B. The leaching of water-soluble nutrients (nitrates, phosphates, simple sugars) from decomposing detritus into the surrounding soil water

C. The complete mineralisation of organic matter by microbial catabolism, releasing inorganic ions such as NH_4^+, PO_4^{3-}, and K^+ into the soil

D. The formation of humus — dark-coloured, chemically complex, recalcitrant organic colloids (polymers of lignin degradation products and microbial biomass) that improve soil structure, water-holding capacity, and nutrient content

Q37. Which set of structural and physiological adaptations is CORRECTLY matched to organisms living in the **desert biome**?

A. Plants: CAM metabolism (stomata open at night for CO_2 fixation), succulent stem/leaf tissues for water storage, spines (modified leaves to reduce transpiration); Animals: nocturnal activity, production of concentrated urine, aestivation during hot dry periods

B. Plants: broad, thin leaves to maximise light capture in low-light conditions; Animals: thick fur coat for insulation against sub-zero temperatures

C. Plants: buttress roots for anchorage in waterlogged soil, epiphytic growth habits to access canopy light; Animals: arboreal locomotion and frugivory in the rainforest canopy

D. Plants: submerged or floating leaves with abundant aerenchyma (air-filled spaces) for buoyancy; Animals: webbed feet for swimming, gill slits in larval stages

Q38. A pollution control board measures the water quality of a river receiving untreated sewage discharge from a town. The board reports a BOD (Biological Oxygen Demand) value of 45 mg/L at the discharge point, compared to 1 mg/L 5 km upstream. What does the high BOD value indicate, and what ecological consequence follows?

A. A high BOD indicates high dissolved oxygen content and a healthy population of aerobic microorganisms maintaining good water quality

B. A high BOD indicates high organic pollution — heterotrophic microorganisms consume large amounts of dissolved oxygen while decomposing the organic matter, leading to hypoxia that harms or kills aerobic aquatic organisms such as fish

C. A high BOD reflects intense photosynthetic activity by phytoplankton, which releases oxygen during the day and increases BOD measurements

D. A high BOD indicates very low numbers of heterotrophic bacteria, so organic matter accumulates undecomposed and oxygen is unused

Q39. CRISPR-Cas9 is a bacterial adaptive immune-derived genome editing tool now widely used in research and medicine. What is the specific molecular role of the guide RNA (gRNA) in this system?

A. The gRNA functions as an independent endonuclease that cleaves double-stranded DNA at a specific target sequence without requiring the Cas9 protein

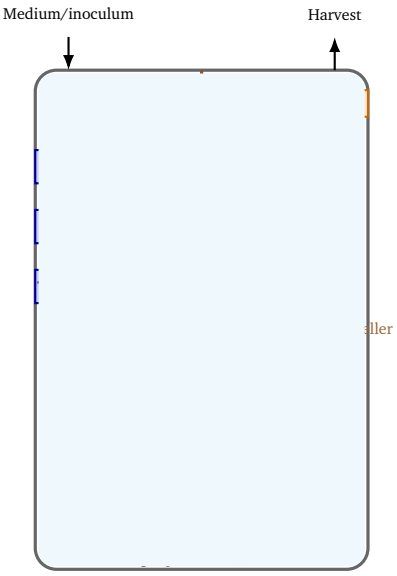
B. The gRNA encodes the Cas9 protein sequence; after translation, Cas9 localises to the nucleus and scans DNA for its target

C. The gRNA (comprising the CRISPR RNA + tracrRNA scaffold) directs the Cas9 endonuclease to a specific genomic locus by base-pairing its 20-nucleotide spacer sequence with the complementary target DNA strand, immediately upstream of a PAM sequence (5′-NGG-3′ for *S. pyogenes* Cas9), where Cas9 makes a blunt-ended double-strand break

D. The gRNA acts as a repair template that, after Cas9 cuts the DNA, provides the homologous sequence for high-fidelity repair by the HDR pathway

Q40. The diagram below shows a stirred-tank bioreactor used for large-scale

production of biological products.



The impeller in a stirred-tank bioreactor serves which primary function?

A. To filter incoming air and sterilise it before it enters the culture medium through the sparger

B. To create a zone of positive pressure at the top of the vessel, maintaining sterility by preventing contaminant ingress

C. To continuously harvest the biological product from the culture medium without disturbing cell growth

D. To agitate the culture medium, ensuring uniform distribution of nutrients, cells, pH, and dissolved gases (O_2 and CO_2), thereby enhancing mass transfer and maintaining homogenous culture conditions

Detailed Solutions

Q1.

Solution

Concept — Brush-Border Disaccharidases (Succus Entericus)

Sucrase-isomaltase is a bifunctional enzyme anchored in the brush border membrane (microvillous surface) of small intestinal enterocytes. Its sucrase activity hydrolyses sucrose into glucose and fructose; its isomaltase activity cleaves α-1,6-glycosidic bonds in limit dextrins. These monosaccharides are then absorbed via SGLT1 (glucose/galactose) and GLUT5 (fructose) transporters. Deficiency of sucrase-isomaltase causes osmotic diarrhoea after sucrose ingestion.

Step 1 — Localisation:

Unlike salivary and pancreatic amylases, which are secreted *into* the lumen, sucrase is an integral membrane protein facing the intestinal lumen as part of the glycocalyx of enterocytes.

Step 2 — Specificity:

Sucrase hydrolyses sucrose (α-1,2-glycosidic bond between glucose and fructose); neither salivary nor pancreatic amylase can cleave this bond.

Why other options are wrong:

Option B: Salivary amylase (α-amylase) cleaves α-1,4 bonds in starch, not sucrose, and acts in the oral cavity.

Option C: Pancreatic amylase also cleaves α-1,4 bonds in starch/glycogen and is secreted into the duodenal lumen, but cannot hydrolyse sucrose.

Option D: Sucrase is *not* a pancreatic secretion; the exocrine pancreas does not secrete disaccharidases.

Answer: (A) ← Go back to Q1

Q2.

Solution

Concept — Emulsification and Lipid Digestion

Bile salts are amphipathic molecules (hydrophilic head, hydrophobic tail) synthesised in the liver from cholesterol and secreted into the duodenum via the bile duct. They emulsify large dietary fat globules into micro-scale droplets (micelles), increasing the total surface area for enzymatic attack. Pancreatic lipase then hydrolyses triglycerides at the oil-water interface. Without emulsification, lipase access is severely limited and fat absorption is impaired (steatorrhoea results from bile duct obstruction).

Step 1 — Mechanism:

Bile salts are *not* enzymes; they do not chemically hydrolyse any bond. They are

detergents that mechanically disrupt large fat droplets through the hydrophobic effect, producing a colloidal suspension with dramatically greater surface-area-to-volume ratio.

Step 2 — Consequence:
Greater surface area means more sites for pancreatic lipase (with colipase) to bind and hydrolyse triglycerides into fatty acids and 2-monoglycerides, which are then incorporated into mixed micelles for absorption.

Why other options are wrong:
Option A: Bile salts do not hydrolyse triglycerides; that is the role of pancreatic lipase (and gastric lipase to a minor extent).
Option C: Bile salts do *not* activate pancreatic lipase by conformational change; colipase (a pancreatic co-protein) anchors lipase at the fat droplet surface.
Option D: Bile salts do not convert triglycerides into phospholipids; triglycerides are hydrolysed by lipase to fatty acids and glycerol/monoglycerides.

Answer: (B) ← Go back to Q2

Q3.

Solution

Concept — Dalton's Law of Partial Pressures and Pulmonary Gas Exchange
Dalton's Law states that each gas in a mixture exerts a partial pressure proportional to its mole fraction, independent of other gases. Gases diffuse down their own partial pressure gradients (from higher to lower partial pressure). In the lungs, alveolar $pO_2 \approx 104$ mmHg and venous blood $pO_2 \approx 40$ mmHg — so O_2 diffuses from alveolus into capillary blood. Conversely, venous blood $pCO_2 \approx 45$ mmHg and alveolar $pCO_2 \approx 40$ mmHg — so CO_2 diffuses from blood into alveolus.

Step 1 — O_2 gradient:
$\Delta pO_2 = 104 - 40 = 64$ mmHg driving O_2 from alveolus to blood. Haemoglobin in erythrocytes binds O_2 (oxyhaemoglobin), reducing free dissolved O_2 and maintaining the gradient.

Step 2 — Diffusion, not active transport:
O_2 moves by simple diffusion across the thin alveolar-capillary membrane (type I pneumocytes + fused basement membrane + endothelium, $\approx 0.5\ \mu$m). No ATP is consumed in this process.

Why other options are wrong:
Option A: Haemoglobin binds O_2 passively; it does not "pump" O_2 actively across membranes — it is not a transporter in the membrane.
Option B: Alveolar pCO_2 (40 mmHg) is actually *lower* than venous blood pCO_2 (45 mmHg), so CO_2 moves from blood to alveolus. This is the CO_2 gradient, not

the O_2 gradient.
Option D: Total barometric pressure is the same at both locations; it is the *partial* pressure of individual gases that drives diffusion.

Answer: (C) ← Go back to Q3

Q4.

Solution

Concept — Lung Volumes in Restrictive vs Obstructive Disease
Pulmonary fibrosis is a restrictive lung disease in which progressive scarring reduces lung compliance (the lungs become stiffer). Consequence: the lungs cannot expand fully, so both TLC (total lung capacity) and VC (vital capacity = IRV + TV + ERV) are reduced. In obstructive diseases (e.g., emphysema), the lungs are hyperinflated, TLC and RV increase, but VC may be normal or reduced due to air trapping.

Step 1 — Restrictive pattern:
Spirometry in restrictive disease shows reduced VC, reduced TLC, but the FEV_1/FVC ratio is normal or elevated (because both volumes are equally reduced and air flow is not obstructed). RV may decrease proportionally.

Step 2 — Why fibrosis is restrictive:
Fibrotic tissue replaces elastic lung parenchyma with stiff collagen. This reduces compliance, requiring greater effort to achieve any lung volume, and prevents full inspiration — reducing IRV, TV, ERV, VC, and TLC.

Why other options are wrong:
Option A: RV does not increase in isolation in fibrosis; it tends to decrease along with other volumes.
Option B: FRC may also decrease, but the hallmark of restrictive disease is combined VC + TLC reduction, not isolated FRC reduction.
Option C: An increase in TLC occurs in obstructive (emphysema), *not* restrictive disease; fibrosis causes TLC to decrease.

Answer: (D) ← Go back to Q4

Q5.

Solution

Concept — Primary Haemostasis: Platelet Adhesion via vWF
When a blood vessel wall is damaged, the subendothelial matrix (collagen, fibronectin) is exposed. Von Willebrand factor (vWF), a large multimeric plasma glycoprotein, is adsorbed onto the exposed collagen. The platelet surface glycoprotein complex GPIb/IX/V then binds vWF, mediating initial platelet adhesion —

this is the first step of the platelet plug (primary haemostasis). vWF also carries and stabilises coagulation factor VIII in plasma.

Step 1 — Adhesion cascade:
Exposed collagen binds circulating vWF → GPIb/IX on platelet surface binds vWF → platelet slows and adheres → platelet activation → release of ADP, TXA_2 → platelet aggregation via GPIIb/IIIa binding fibrinogen → platelet plug.

Step 2 — Clinical relevance:
Deficiency of vWF causes von Willebrand disease (most common inherited bleeding disorder), characterised by prolonged bleeding time, mucosal bleeding, and menorrhagia.

Why other options are wrong:
Option B: Factor X activation is part of the coagulation (secondary haemostasis) cascade, not a vWF function.
Option C: Fibrin is derived from fibrinogen by thrombin cleavage; vWF does not synthesise fibrin.
Option D: Thrombopoietin is produced by the liver and kidneys in response to low platelet count and acts on megakaryocytes; it has no relationship with vWF.

Answer: (A) ← Go back to Q5

Q6.

Solution

Concept — Starling Forces and Oedema Formation
Ernest Starling described four forces governing transcapillary fluid movement: capillary hydrostatic pressure (Pc, outward), plasma oncotic pressure (πc, inward), interstitial hydrostatic pressure (Pi, usually slightly inward/negative), and interstitial oncotic pressure (πi, outward). Net filtration pressure = (Pc + πi) − (πc + Pi). Excess fluid not reabsorbed is drained by lymphatics. Oedema forms when net filtration exceeds lymphatic drainage.

Step 1 — Causes of oedema:
(i) Decreased πc (hypoalbuminaemia — liver disease, nephrotic syndrome, malnutrition) → less inward pull → net filtration throughout capillary. (ii) Increased Pc (right heart failure, venous obstruction) → greater outward push → excess filtration. (iii) Lymphatic obstruction (elephantiasis) → fluid not drained.

Step 2 — Normal vs oedematous state:
Normally, small excess filtrate is returned to circulation via lymphatics. When Starling forces are disrupted (especially πc falls or Pc rises), lymphatics are overwhelmed and interstitial fluid accumulates as oedema.

Why other options are wrong:

Option A: Decreased Pc reduces filtration and thus *prevents* oedema; it would cause dehydration of tissues, not oedema.
Option C: Increased lymphatic flow actually *prevents* oedema by removing excess interstitial fluid more efficiently.
Option D: Falling arterial blood pressure reduces capillary filtration, leading to reduced interstitial fluid, not oedema.

Answer: (B) ← Go back to Q6

Q7.

Solution

Concept — Baroreceptor Reflex (Baroreflex)
The baroreflex is a negative feedback mechanism that provides rapid (within seconds) regulation of arterial blood pressure. Stretch-sensitive baroreceptors in the carotid sinus (innervated by CN IX, glossopharyngeal) and aortic arch (innervated by CN X, vagus) detect changes in wall tension proportional to arterial pressure. Afferent signals travel to the nucleus tractus solitarius (NTS) in the medulla, which coordinates the cardiovascular control centre.

Step 1 — Response to rising BP:
Increased wall stretch → increased baroreceptor firing rate → NTS activates vagal (parasympathetic) output to SA node (decreasing HR) and inhibits sympathetic outflow to heart and arterioles (decreasing contractility and peripheral resistance) → BP falls back toward normal.

Step 2 — Response to falling BP:
Decreased stretch → decreased baroreceptor firing → reduced vagal activity → increased sympathetic activity → increased HR, stroke volume, and vasoconstriction → BP rises.

Why other options are wrong:
Option A: Baroreceptors are in the carotid sinus and aortic arch, not the left ventricular wall (ventricular mechanoreceptors exist but do not mediate the classical baroreflex).
Option B: Adrenal medulla epinephrine release is a slower, sympathetically mediated response; baroreceptors primarily regulate autonomic tone, not adrenal secretion directly.
Option D: Renin release is part of the slower RAAS mechanism for volume/BP regulation; baroreceptors act via rapid autonomic mechanisms, not primarily via renin.

Answer: (C) ← Go back to Q7

Q8.

Solution

Concept — Nephrolithiasis: Composition and Epidemiology

Kidney stones form when urine becomes supersaturated with crystallogenic substances. Epidemiologically, calcium oxalate (often mixed with calcium phosphate) accounts for approximately 70–80% of all urinary tract stones in Western populations. Calcium oxalate stones are radio-opaque on plain X-ray. Key risk factors include hypercalciuria (excess Ca^{2+} in urine), hyperoxaluria (excess oxalate, from dietary sources or primary hyperoxaluria), dehydration, and low urinary citrate (a natural inhibitor of crystallisation).

Step 1 — Other stone types (less common):

Uric acid stones ($\approx$5–10%) are radiolucent and occur in gout or high-purine diets. Struvite (magnesium ammonium phosphate) stones ($\approx$5–15%) are caused by urease-positive bacteria (e.g., *Proteus mirabilis*). Cystine stones ($<$3%) result from cystinuria, a rare autosomal recessive disorder of renal amino acid transport.

Step 2 — Treatment rationale:

Calcium oxalate stones: increased hydration, dietary calcium normalisation (paradoxically, very low calcium diet increases oxalate absorption), thiazide diuretics (reduce urinary Ca^{2+}), citrate supplementation.

Why other options are wrong:

Option A: Uric acid stones are the second-most-common type in some populations but overall represent only $\approx$5–10% of stones.

Option B: Struvite stones are always infection-associated (urease-positive bacteria); they are not the most common type.

Option C: Cystine stones are rare ($<$3%) and confined to patients with the inherited cystinuria disorder.

Answer: (D) ← Go back to Q8

Q9.

Solution

Concept — Osmoregulation in Marine Teleost Fish (Hyper-regulator challenge)

Seawater osmolarity ($\approx$1000 mOsm/kg) is much higher than marine teleost body fluids ($\approx$300–400 mOsm/kg). This osmotic gradient causes the fish to lose water by osmosis through gills and body surfaces (they are hypoosmotic relative to seawater). To compensate for this water loss, marine teleosts drink seawater continuously (up to 20% body weight per day).

Step 1 — Salt excretion:

The ingested seawater provides water but also delivers a large NaCl load. Excess Na^+ and Cl^- are actively excreted across gill ionocytes (mitochondria-rich chloride

cells) by Na^+/K^+-ATPase and chloride channels (CFTR-like). The kidneys produce small volumes of nearly isotonic urine (teleost kidneys have few glomeruli and limited concentrating ability compared to mammals).

Step 2 — Contrast with freshwater fish:
Freshwater teleosts are hyperosmotic to their environment; they gain water by osmosis (never drink), produce copious dilute urine, and actively absorb ions from the water via gill ionocytes.

Why other options are wrong:
Option B: The goal is *not* to match internal osmolarity to seawater; the fish actively regulates to maintain a much lower internal osmolarity than seawater.
Option C: Marine teleosts produce *small* volumes of concentrated urine, not large volumes of dilute urine (that is the freshwater fish strategy).
Option D: Passive ion absorption by simple diffusion would worsen salt loading; ion transport across gills is active and directional (excretion, not absorption, of Na^+ and Cl^-).

Answer: (A) ← Go back to Q9

Q10.

Solution

Concept — Counter-Current Multiplier: Loop of Henle and Medullary Gradient
The loop of Henle functions as a counter-current multiplier to establish a steep osmotic gradient in the renal medulla (from ≈300 mOsm/kg in the cortex to ≈1200 mOsm/kg in the inner medulla). The thin descending limb is permeable to water but not to NaCl: water exits by osmosis, concentrating the filtrate. The thick ascending limb (TAL) actively transports NaCl (via NKCC2 co-transporter) out of the tubule and into the interstitium but is impermeable to water, diluting the filtrate.

Step 1 — Role of ADH:
The hypertonic medullary interstitium created by the loop is utilised by the collecting duct: when ADH (vasopressin, from posterior pituitary) is present, aquaporin-2 (AQP2) channels are inserted into collecting duct principal cells, allowing water reabsorption down the osmotic gradient into the hypertonic interstitium, concentrating the urine.

Step 2 — Vasa recta:
The vasa recta (hairpin-shaped capillaries paralleling the loop) act as counter-current exchangers that passively maintain the medullary gradient without washing it away.

Why other options are wrong:

Option A: The ascending limb dilutes (not concentrates by adding water) the filtrate by pumping *salt out*, not by adding water in. Water cannot enter the ascending limb.
Option C: Glucose reabsorption occurs in the *proximal* convoluted tubule via SGLT2/SGLT1 transporters, not in the loop of Henle.
Option D: H^+ and K^+ secretion occurs mainly in the collecting duct principal and intercalated cells, not the loop of Henle.

Answer: (B) ← Go back to Q10

Q11.

Solution

Concept — Muscle Twitch Summation and Tetanic Contraction
A single twitch is the mechanical response to one action potential: Ca^{2+} is released from the SR, binds troponin, myosin cross-bridges cycle, and then Ca^{2+} is resequestered. If a second stimulus arrives before relaxation is complete, the second twitch builds on residual Ca^{2+} and cross-bridge activation (wave summation). At high stimulation frequencies, Ca^{2+} accumulates in the cytoplasm because SR reuptake cannot keep pace, maintaining maximum troponin-C saturation and maximal cross-bridge cycling — this is complete (fused) tetanus.

Step 1 — Complete tetanus:
At frequencies above the tetanic fusion frequency (typically 50–100 Hz in fast-twitch fibres, lower in slow-twitch), successive twitches merge into a smooth, sustained plateau of maximum force. There is no relaxation between stimuli.

Step 2 — Importance:
Most voluntary movements in life are produced by graded, partly or fully tetanic contractions, not isolated twitches. Motor unit recruitment and rate coding are the two mechanisms for grading muscle force.

Why other options are wrong:
Option A: If each twitch fully relaxes before the next stimulus, no summation occurs and forces remain at single-twitch levels — this describes *unfused* or *isolated* twitches, not tetanus.
Option B: Fatigue causes *decline* in force (reduced ATP, lactic acid, Ca^{2+} depletion), not the sustained maximal force of complete tetanus.
Option D: Complete SR Ca^{2+} depletion would prevent contraction entirely (rigor or paralysis), not a sustained maximum force plateau.

Answer: (C) ← Go back to Q11

Q12.

Solution

Concept — Muscle Fibre Types: Type I (Slow-Oxidative) vs Type IIb (Fast-Glycolytic)

Skeletal muscle fibres are classified by their myosin heavy chain isoform and metabolic profile. Type I (slow-twitch, SO) fibres: contain abundant myoglobin (red colour), high mitochondrial density, rich capillary supply, predominantly use oxidative phosphorylation, generate moderate force but sustain it for hours without fatigue. Type IIb (fast-twitch, FG) fibres: low myoglobin (white), few mitochondria, high glycolytic enzyme activity, generate high peak force rapidly but fatigue within minutes due to lactate accumulation.

Step 1 — Marathon requirement:

A marathon (42.195 km) requires sustained aerobic power output for 2–5 hours. Type I fibres' high oxidative capacity and fatigue resistance make them ideal. Type IIb fibres are exhausted within minutes and cannot sustain aerobic power at marathon pace.

Step 2 — Adaptation:

Trained endurance athletes have a higher proportion of Type I fibres and increased mitochondrial density (mitochondrial biogenesis via PGC-1α) in their locomotor muscles.

Why other options are wrong:

Option A: Type IIb fast-twitch fibres are used for explosive activities (100 m sprint, weightlifting) where maximum power for a few seconds is required, not sustained activity.

Option B: Type IIa (fast-oxidative-glycolytic) fibres are intermediate and used for activities of moderate duration ($\approx$minutes), not a full marathon.

Option C: The reasoning is inverted — slow-twitch fibres are *not* for short bursts but for prolonged endurance; it is fast-twitch fibres that produce short bursts.

Answer: (D) ← Go back to Q12

Q13.

Solution

Concept — Cerebellar Functions and Lesion Signs

The cerebellum does not initiate movement but fine-tunes and coordinates ongoing voluntary movements, maintains balance, and regulates muscle tone. It receives proprioceptive and vestibular input and compares intended vs actual movements. Purkinje cell axons are the sole output of the cerebellar cortex (to deep cerebellar nuclei $\rightarrow$ thalamus $\rightarrow$ motor cortex). Cerebellar lesions produce ipsilateral signs (because the cerebellum controls the same side of the body).

Step 1 — Characteristic cerebellar signs:

(i) Ataxia (wide-based, staggering gait — damage to vestibulocerebellum and spinocerebellum); (ii) Dysmetria/past-pointing (inability to accurately judge distance and stop — finger-to-nose test); (iii) Dysdiadochokinesia (failure of rapid alternating movements — ask patient to rapidly supinate/pronate hand); (iv) Intention tremor (tremor worsens as limb approaches target, contrasted with resting tremor of basal ganglia disease); (v) Nystagmus; (vi) Dysarthria (slurred, scanning speech).

Step 2 — Distinguishing from other lesions:
Basal ganglia disease (Parkinson's): resting tremor, bradykinesia, rigidity, no ataxia. Motor cortex: contralateral weakness (paresis), spasticity, no ataxia. Thalamus: sensory loss, no motor coordination deficits.

Why other options are wrong:
Option B: Primary motor cortex lesion causes contralateral hemiparesis/hemiplegia (upper motor neuron signs: spasticity, hyperreflexia), not cerebellar ataxia or dysmetria.
Option C: Basal ganglia damage causes Parkinson's disease (resting tremor, rigidity, bradykinesia) or Huntington's chorea, not the cerebellar syndrome described.
Option D: Thalamic lesions primarily cause contralateral hemisensory loss (pain, temperature, proprioception) without the specific cerebellar coordination deficits described.

Answer: (A) ← Go back to Q13

Q14.

Solution

Concept — Tonotopic Organisation of the Basilar Membrane (Cochlea)
The basilar membrane runs the length of the cochlear spiral (≈35 mm in humans). Its physical properties vary continuously from base to apex: at the base, it is narrow (≈0.1 mm) and stiff (high stiffness, high resonant frequency, responds to high-frequency sounds); at the apex, it is wide (≈0.5 mm) and floppy (low stiffness, low resonant frequency, responds to low-frequency sounds). This graded mechanical property underlies the tonotopic map of frequency detection.

Step 1 — Place theory:
Von Békésy's "place theory" (Nobel Prize 1961): a given sound frequency creates a travelling wave that reaches maximum amplitude at a specific position on the basilar membrane. Inner hair cells at that position are maximally deflected, opening mechanosensitive stereocilia tip-links, allowing K^+ and Ca^{2+} influx, depolarisation, and release of glutamate onto cochlear nerve fibres.

Step 2 — Neural coding:
Each cochlear nerve fibre has a characteristic frequency (CF) matching the basilar

membrane position from which it receives input. The tonotopic map is preserved all the way to the auditory cortex (primary auditory cortex, Heschl's gyri).

Why other options are wrong:
Option A: This has the frequency mapping inverted. High-frequency sounds are detected at the *base* (stiff, narrow), not the apex; low-frequency sounds at the *apex* (floppy, wide).
Option C: The tympanic membrane vibrates to all frequencies simultaneously and transduces sound energy to the ossicular chain; it does not selectively activate cochlear zones.
Option D: The ossicles transmit sound from tympanic membrane to oval window amplifying force (due to area ratio); they transmit all frequencies and do not filter by frequency.

Answer: (B) ← Go back to Q14

Q15.

Solution

Concept — Gonadotrophins: FSH and LH Target Cells in the Testis
The two gonadotrophins (FSH and LH) from anterior pituitary gonadotrophs act on distinct cell populations in the testis. The testis contains two key somatic cell types: Sertoli cells (within seminiferous tubules) and Leydig (interstitial) cells (between tubules). FSH receptors are expressed on Sertoli cells; LH (which in males is sometimes called ICSH — interstitial cell-stimulating hormone) receptors are expressed on Leydig cells.

Step 1 — FSH on Sertoli cells:
FSH binding to Sertoli cells stimulates production of androgen-binding protein (ABP, which concentrates testosterone in the tubular lumen), inhibin B (negative feedback on FSH), and various growth factors that support spermatogonia proliferation, meiosis, and spermiogenesis. Sertoli cells form the blood-testis barrier.

Step 2 — LH (ICSH) on Leydig cells:
LH binds Leydig cell receptors, activating adenylyl cyclase → cAMP → PKA → StAR protein → cholesterol transport into mitochondria → testosterone synthesis (steroidogenesis). Testosterone acts locally (high concentrations in tubules via ABP) to support spermatogenesis and systemically for secondary sexual characteristics.

Why other options are wrong:
Option A: FSH targets Sertoli cells, not Leydig cells; LH targets Leydig cells for testosterone — this option has FSH and Leydig cell incorrectly paired.
Option B: LH targets Leydig cells (not Sertoli cells) for testosterone; FSH acts on Sertoli cells for spermatogenesis support — this option has LH and Sertoli cell

incorrectly paired.
Option D: FSH and LH do not both target Leydig cells; they target distinct cell populations (Sertoli and Leydig respectively).

Answer: (C) ← Go back to Q15

Q16.

Solution

Concept — Oxytocin and Positive Feedback During Parturition (Ferguson Reflex)
Oxytocin (a 9-amino-acid peptide) is synthesised in the hypothalamic paraventricular nucleus (PVN) and supraoptic nucleus and released from the posterior pituitary (neurohypophysis). During parturition, the fetal presenting part (usually the head) presses against the cervix, activating mechanoreceptors (stretch receptors) in the cervix and lower uterus. Afferent signals travel via the spinal cord to the hypothalamus → oxytocin release from posterior pituitary → uterine contractions (oxytocin binds OXTR, a Gq-coupled GPCR on myometrium).

Step 1 — Positive feedback loop:
Contractions push the fetus against the cervix with greater force → more cervical stretching → more afferent signals to hypothalamus → more oxytocin release → stronger, more frequent contractions. This escalating positive feedback continues until the baby (and placenta) is fully expelled, eliminating the cervical stretch stimulus.

Step 2 — Second function:
After birth, suckling stimulates nipple mechanoreceptors → hypothalamus → posterior pituitary → oxytocin → milk ejection (let-down reflex) by contracting myoepithelial cells around alveoli.

Why other options are wrong:
Option A: Oxytocin does not stimulate oestrogen synthesis in the corpus luteum; the corpus luteum has already become the corpus albicans by late pregnancy, and the placenta produces oestrogen.
Option B: Progesterone withdrawal (through changes in receptor isoforms) does contribute to labour onset, but this is a separate mechanism, not how oxytocin creates positive feedback.
Option C: Oxytocin is not secreted at a fixed constant rate; its release is pulsatile and stimulus-triggered (cervical stretch or suckling).

Answer: (D) ← Go back to Q16

Q17.

Solution

Concept — LDL Receptor Pathway and Receptor Recycling

The LDL receptor (LDLR) pathway, elucidated by Goldstein and Brown (Nobel Prize 1985), is the paradigm for receptor-mediated endocytosis. LDL particles bind LDLR at coated pits (clathrin-coated regions of the plasma membrane). The pit invaginates and pinches off as a clathrin-coated vesicle $\rightarrow$ clathrin coat is shed $\rightarrow$ early endosome (pH $\approx$ 6.2). The acidic pH causes LDL to dissociate from LDLR. The receptor is then sorted into recycling vesicles (tubular extensions of the early endosome) and returns to the plasma membrane, while the LDL particle is delivered to the late endosome and then the lysosome.

Step 1 — Lysosomal processing of LDL:

In the lysosome (pH $\approx$ 4.8), acid hydrolases degrade the apoB-100 protein, and cholesterol esters are hydrolysed by acid lipase to free cholesterol. Free cholesterol regulates intracellular cholesterol homeostasis (suppresses HMG-CoA reductase, activates ACAT for cholesterol esterification, downregulates LDLR expression).

Step 2 — Efficiency of recycling:

Each LDLR makes approximately 150–200 trips between the cell surface and endosomes during its lifetime ($\approx$20 hours). This recycling is energetically efficient and allows each receptor to deliver many LDL particles.

Why other options are wrong:

Option B: The receptor is *not* degraded in the lysosome under normal conditions; only the LDL cargo is. Degradation of LDLR occurs only in pathological conditions or after prolonged lifespan.

Option C: Receptors are not transported to the Golgi for permanent storage; newly synthesised receptors come from the Golgi, but recycled receptors go directly from early endosomes back to the plasma membrane.

Option D: Receptors are not exported by exocytosis; exocytosis involves fusion of vesicles from the secretory pathway, not recycling endosomes.

Answer: (A) ← Go back to Q17

Q18.

Solution

Concept — Apoptosis vs Necrosis: Morphological and Molecular Distinctions

Apoptosis (Greek: "falling away of leaves") is a programmed, energy-dependent, caspase-mediated process of cell death essential for development (e.g., digit separation, thymic T-cell selection) and tissue homeostasis. Key morphological features: cell shrinkage, chromatin condensation (pyknosis) and fragmentation (karyorrhexis), membrane blebbing, and fragmentation into membrane-bound apoptotic bodies that are phagocytosed by macrophages or neighbouring cells.

No inflammation occurs because membrane integrity is maintained until phagocytosis.

Step 1 — Caspase cascade:
Intrinsic pathway: cytochrome c + APAF-1 + caspase 9 → apoptosome → executioner caspases (3, 6, 7) → PARP cleavage, CAD-mediated DNA ladder (180 bp multimers), cytoskeletal fragmentation. Extrinsic pathway: Fas/FasL, TNF-R → DISC → caspase 8 → executioner caspases.

Step 2 — Necrosis contrast:
Necrosis (now also called NCID — necrotic cell death): passive, energy-independent cell swelling (oncosis), organelle swelling, membrane rupture, release of DAMPs (damage-associated molecular patterns), triggering an inflammatory response. No caspase involvement in classical necrosis.

Why other options are wrong:
Option A: Cell swelling, membrane rupture, and inflammation are features of *necrosis*, not apoptosis. The question presents a case of apoptosis (shrinkage, condensed chromatin, no inflammation).
Option C: Necrosis is *not* energy-dependent or caspase-mediated; it is a passive, unregulated process (though regulated necrosis — necroptosis — exists, it is distinct from classical necrosis).
Option D: Apoptosis can be triggered by both extrinsic (death receptor, immune cell-mediated) and intrinsic (DNA damage, oxidative stress, hypoxia) pathways; DNA damage is a classical intrinsic trigger.

Answer: (B) ← Go back to Q18

Q19.

Solution

Concept — Essential Features of a Plasmid Cloning Vector
A plasmid cloning vector must possess three indispensable components: (1) An **origin of replication (ori)** — without it the plasmid cannot replicate autonomously in the bacterial host and will be diluted out after cell division. Different ori sequences determine copy number (high-copy: pUC series ≈500 copies/cell; low-copy: pACYC ≈5–10). (2) A **selectable marker** — typically an antibiotic resistance gene (e.g., Amp^R, Kan^R, Tet^R) — allows identification and selection of bacteria that have successfully taken up the plasmid. Without selection, transformants are rapidly outcompeted. (3) A **multiple cloning site (MCS)** — a short polylinker sequence containing many unique restriction enzyme recognition sites clustered together, allowing insertion of foreign DNA using various restriction enzymes.

Step 1 — Optional but useful features:

lacZ gene fragment (for blue-white screening to identify recombinants), promoters for in vitro transcription, f1 phage origin for ssDNA production, etc.

Step 2 — Prokaryotic vs eukaryotic requirements:
Bacteria lack a nucleus, chromosomal centromeres, and histones; plasmid replication in bacteria does not require any of these eukaryotic features.

Why other options are wrong:
Option A: Centromeres, telomeres, and histones are features of *eukaryotic* chromosomes. Bacteria have neither a nucleus nor a mitotic apparatus requiring centromeres or telomeres; plasmids replicate extrachromosomally without these.
Option B: An antibiotic resistance gene alone is insufficient; without an ori, the plasmid cannot replicate and will be lost; without an MCS, foreign DNA cannot be inserted.
Option D: Bacteria do not have a nuclear membrane; plasmid DNA is directly accessible to cytoplasmic machinery, and bacterial nucleases are not a significant impediment to maintained plasmid function.

Answer: (C) ← Go back to Q19

Q20.

Solution

Concept — Founder Effect: A Special Case of Genetic Drift
Genetic drift refers to random changes in allele frequencies in populations due to chance (sampling error), not selection. The founder effect occurs when a small number of individuals from a large source population colonise a new, isolated habitat (island, forest fragment, valley). The founders carry only a random, possibly non-representative subset of the source population's allele pool, so the new population has: (i) lower overall genetic diversity (fewer alleles), (ii) allele frequencies that differ from the source population by chance, and (iii) possibly higher frequencies of rare alleles if a founder happened to carry them.

Step 1 — Classic examples:
Amish community (USA): high frequency of Ellis-van Creveld syndrome (rare dwarfism) due to founder effect from a small group of 18th-century German immigrants. Pacific island human populations: reduced mitochondrial DNA diversity compared to mainland populations. Darwin's finches on Galápagos: founded by a small group from South America.

Step 2 — Distinction from bottleneck:
The bottleneck effect also involves a drastic reduction in population size, but the survivors are remnants of an *existing* large population after a catastrophe (disease, hunting, natural disaster), rather than colonisers of a new location.

Why other options are wrong:
Option A: This describes the *bottleneck* effect (reduction of an existing population by catastrophe), not the founder effect.
Option B: Natural selection is a non-random, fitness-based process, not a form of genetic drift; the founder effect is random and non-adaptive.
Option C: Gene flow involves migration *into* the population from outside, which increases genetic diversity — the opposite of the founder effect.

Answer: (D) ← Go back to Q**20**

Q21.

Solution

Concept — Analogous Structures and Convergent Evolution
Analogous structures are those that perform a similar function but have fundamentally different evolutionary origins and internal structural blueprints. They arise independently in unrelated lineages through convergent evolution, where similar ecological pressures (e.g., the selective advantage of powered flight) drive different ancestral structures toward a similar functional solution. In contrast, homologous structures share a common embryological origin (even if they serve different functions today) and reveal common ancestry.

Step 1 — Wings compared:
Bird wing: modified forelimb (radius, ulna, humerus + fused carpometacarpus) with feathers evolved from scales. Bat wing: also a forelimb (same bones), but with greatly elongated digit bones supporting a wing membrane (patagium). Insect wing: cuticular outgrowth of the thorax (not a forelimb at all; no homology with vertebrate limbs). Bird and bat wings are actually homologous to each other (both are modified tetrapod forelimbs) but are analogous to insect wings.

Step 2 — Convergent evolution:
Independent evolution of wing-like structures has also occurred in pterosaurs, flying squirrels, flying fish, and sugar gliders — all are analogous to each other and to insect wings in functional terms.

Why other options are wrong:
Option B: Bird and bat wings share the same basic forelimb developmental pathway (they are homologous to each other), but insect wings do not share this pathway — insect wings are cuticular structures unrelated to vertebrate forelimbs.
Option C: Insect wings have no internal bones whatsoever; they consist of veined cuticular outgrowths. The wing bone arrangement (humerus, radius, ulna) is shared only between birds, bats, and other tetrapod forelimbs.
Option D: There was no shared ancestral wing in early tetrapods (amphibians) — tetrapod forelimbs evolved into wings independently in birds, bats, and

pterosaurs; insects diverged from vertebrates far earlier (before any tetrapod existed).

Answer: (A) ← Go back to Q21

Q22.

Solution

Concept — Chi-Square Test in Genetics: Testing Mendelian Ratios

The chi-square (χ^2) goodness-of-fit test evaluates whether observed frequencies differ from expected frequencies by more than random chance alone. The null hypothesis (H_0): the observed data fit the expected 3:1 Mendelian ratio. $\chi^2 = \sum \frac{(O-E)^2}{E}$. For this cross: $\chi^2 = \frac{(80-75)^2}{75} + \frac{(20-25)^2}{25} = \frac{25}{75} + \frac{25}{25} = 0.33 + 1.00 = 1.33$.

Step 1 — Interpretation:

Degrees of freedom (df) = number of phenotypic classes $-$ 1 = 2 $-$ 1 = 1. At df = 1 and p = 0.05 significance level, the critical χ^2 value is 3.841. Since 1.33 $<$ 3.841, we *fail to reject* H_0. The deviation from the expected 3:1 ratio is within the range expected by random sampling variation.

Step 2 — Biological conclusion:

The observed 80:20 ratio is consistent with a standard 3:1 Mendelian ratio for a monohybrid cross ($Aa \times Aa$). There is no statistical evidence to reject simple dominance inheritance.

Why other options are wrong:

Option A: The χ^2 value (1.33) is well below the critical value (3.84), so the result is *not* statistically significant; we cannot reject the 3:1 hypothesis.

Option C: Statistical significance depends on the magnitude of deviation relative to the critical value, not merely on whether observed counts exceed expected counts. A slightly larger observed dominant class is expected by chance in 100 trials.

Option D: There is no minimum sample size rule of 500 for applying the chi-square test; in practice, the test requires expected frequencies of at least 5 per class, which is met here (75 and 25).

Answer: (B) ← Go back to Q22

Q23.

Solution

Concept — Cytoplasmic (Maternal) Inheritance and Reciprocal Cross Analysis

Cytoplasmic (extranuclear) inheritance involves traits encoded by organellar genomes — mitochondrial DNA (mtDNA) or chloroplast DNA (cpDNA). Because egg cells contribute essentially all of the cytoplasm (and hence organelles) to

the zygote, while sperm contribute only nuclear DNA, the offspring's cytoplasmic genotype is determined by the maternal parent, not the paternal. This leads to a distinctive pattern: reciprocal crosses give *different* results because the maternal parent's identity changes.

Step 1 — Classic example:
Variegation in *Mirabilis jalapa* (four o'clock plant) is caused by plastid (chloroplast) mutations affecting photosynthetic pigmentation. Maternal plastids are inherited, so only the mother's plastid genotype determines the offspring's leaf colour. In nuclear inheritance, both reciprocal crosses would give the same F_1 phenotype (regardless of which parent is male/female for autosomal traits).

Step 2 — Mitochondrial inheritance in animals:
In animals, mtDNA is strictly maternally inherited (LHON, mitochondrial myopathies, MELAS). All offspring of an affected mother inherit the mutation; no offspring of an affected father inherit the condition.

Why other options are wrong:
Option A: Identical results in reciprocal crosses is the hallmark of *autosomal* (nuclear) Mendelian inheritance, where both parents contribute equally to the nuclear genome of each offspring.
Option B: Paternal (cytoplasmic) inheritance would mean offspring follow the father's phenotype; this is extremely rare (some exceptions in mussels). Maternal inheritance is the rule for most organellar traits.
Option D: A 3:1 F_2 ratio is the signature of nuclear Mendelian monohybrid inheritance with complete dominance, not cytoplasmic inheritance.

Answer: (C) ← Go back to Q23

Q24.

Solution

Concept — Telomerase, Telomere Maintenance, and Cancer
Telomeres are repetitive hexanucleotide sequences (5′-TTAGGG-3′) that cap chromosomal ends, preventing degradation and end-to-end chromosome fusions. Each round of DNA replication shortens telomeres by 50–200 bp (the "end-replication problem") because DNA polymerase cannot replicate the very terminus of the lagging strand. In most somatic cells, telomerase is absent or at very low levels; telomeres shorten with each division until a critical length triggers replicative senescence (Hayflick limit, ≈50–70 divisions) or apoptosis.

Step 1 — Telomerase in cancer:
Approximately 85–90% of human cancers express high levels of telomerase (by reactivating the catalytic subunit TERT gene, often through promoter mutations). This allows cancer cells to maintain telomere length indefinitely, circumventing

the Hayflick limit and achieving replicative immortality (one of Hanahan and Weinberg's hallmarks of cancer).

Step 2 — Therapeutic rationale:
Inhibiting telomerase (e.g., with imetelstat, a telomerase template antagonist) would progressively shorten telomeres in cancer cells across successive divisions, eventually triggering senescence or apoptosis. Normal somatic cells with inactive telomerase would be minimally affected.

Why other options are wrong:
Option A: Telomerase is a specialised reverse transcriptase that extends telomeric ends using its intrinsic RNA template (TERC); it does not participate in normal S-phase replication at origins or in mismatch repair.
Option B: Telomerase extends only single-stranded telomeric overhangs; it has no function in double-strand break repair at non-telomeric sites.
Option C: Normal somatic cells (skin fibroblasts, hepatocytes, cardiomyocytes) have negligible or zero telomerase activity. Germ cells, stem cells, and activated lymphocytes have measurable telomerase. This makes telomerase a selective target for cancer cells, but the premise that "all somatic cells express telomerase" is incorrect.

Answer: (D) ← Go back to Q24

Q25.

Solution

Concept — Post-Translational Modifications (PTMs) and Their Functions
After the primary polypeptide is synthesised by the ribosome, numerous covalent modifications can alter its properties. The most biologically ubiquitous and significant PTM is phosphorylation: protein kinases (serine/threonine kinases, tyrosine kinases) covalently attach a phosphate group from ATP to hydroxyl groups of Ser, Thr, or Tyr residues. Phosphorylation alters the local charge (-2 at physiological pH) and conformation, which can activate, inactivate, or redirect protein-protein interactions. Protein phosphatases reverse the modification. This "phosphoswitch" is the central mechanism of intracellular signal transduction (e.g., MAP kinase cascade, insulin signalling via PI3K/Akt, cAMP/PKA pathway).

Step 1 — Examples of phosphorylation as signal:
Epinephrine $\rightarrow$ cAMP $\rightarrow$ PKA $\rightarrow$ phosphorylates glycogen phosphorylase kinase (activates it) $\rightarrow$ glycogenolysis. EGF receptor autophosphorylation on Tyr $\rightarrow$ Ras/MAPK cascade $\rightarrow$ cell proliferation.

Step 2 — Other major PTMs:
Ubiquitination (polyUb chain $\rightarrow$ 26S proteasome degradation), glycosylation (N-linked in ER, O-linked in Golgi), acetylation (histone tails, regulatory proteins),

and proteolytic cleavage (zymogens: proinsulin → insulin; procollagen → collagen).

Why other options are wrong:
Option B: Translation of mRNA to polypeptide is the process of *synthesis*, not a post-translational modification; it occurs before any PTM.
Option C: Removal of introns from pre-mRNA is RNA splicing, a *post-transcriptional* (not post-translational) modification occurring in the nucleus before the mRNA is translated.
Option D: The 5′ 7-methylguanosine cap is added to pre-mRNA in the nucleus, immediately after transcription begins; it is a *post-transcriptional* RNA modification, not a protein PTM.

Answer: (A) ← Go back to Q25

Q26.

Solution

Concept — Histone Acetylation, Chromatin Structure, and Transcriptional Activation
Nucleosomes consist of 147 bp of DNA wrapped ≈1.65 times around a histone octamer (H2A, H2B, H3, H4, two copies each). Histone tails (especially H3 and H4) protrude from the nucleosome and are rich in positively charged lysine (Lys, $+1$ at pH 7) and arginine residues that interact electrostatically with the negatively charged phosphate backbone of DNA.

Step 1 — Effect of acetylation:
Histone acetyltransferases (HATs, e.g., CBP/p300, PCAF) transfer an acetyl group (CH_3CO-) from acetyl-CoA to the ε-amino group of lysine, neutralising its positive charge. This reduces electrostatic attraction between histone tails and DNA → nucleosome spacing increases → chromatin "opens" into a more accessible euchromatic conformation → transcription factors and RNA Pol II can access promoters and enhancers.

Step 2 — Acetyl-lysine as a landing pad:
Beyond charge neutralisation, acetylated lysine is recognised by bromodomain-containing proteins (e.g., in TAFs, SWI/SNF) that further remodel chromatin and assemble the pre-initiation complex.

Why other options are wrong:
Option A: Acetylation does not remove histones from the nucleosome; the histone octamer remains associated with DNA. Histone eviction requires specialised remodelling complexes (e.g., SWI/SNF) and is not caused by acetylation directly.
Option C: Acetylation adds small two-carbon acetyl groups (not "bulky carbohydrate groups"). Carbohydrate additions are made by glycosyltransferases (O-

GlcNAc modification), a completely different PTM.
Option D: Histone acetylation and DNA/promoter methylation are separate epigenetic layers; HATs do not methylate DNA. CpG methylation is carried out by DNA methyltransferases (DNMTs), generally associated with gene *silencing* rather than activation.

Answer: (B) ← Go back to Q**26**

Q27.

Solution

Concept — Ethylene: Triple Response in Etiolated Dicot Seedlings
Ethylene ($CH_2{=}CH_2$) is a gaseous plant hormone detected by ETR1/ETR2 receptor kinases in the endoplasmic reticulum membrane. When etiolated (dark-grown) dicot seedlings encounter a physical obstacle during germination, local ethylene synthesis increases. The "triple response" is the resulting phenotype: (1) inhibition of hypocotyl elongation (shorter stem), (2) radial swelling of the hypocotyl (thicker stem), and (3) exaggeration and maintenance of the apical hook (protective curved tip). This response is believed to help the seedling push through or around soil obstacles.

Step 1 — Molecular basis:
Without ethylene: ETR1 constitutively inhibits CTR1 → CTR1 is inactive → EIN2/EIN3/EIL1 active → normal elongation gene programme. With ethylene: ETR1 inactivated → CTR1 no longer represses EIN2 → EIN2 C-terminal is cleaved → EIN3/EIL1 transcription factors active → ethylene-responsive genes expressed (including short-hypocotyl, hook maintenance).

Step 2 — Other roles of ethylene:
Fruit ripening (climacteric fruits: apple, banana, mango) via positive autocatalytic ethylene synthesis (System 2: ethylene induces more ethylene). Abscission (separation layer at petiole/petiolule base). Flooding response (aerenchyma formation via cortical cell lysigenous PCD). Flower senescence.

Why other options are wrong:
Option A: Ethylene *inhibits* elongation (shorter hypocotyl) and *exaggerates* (does not close/straighten) the apical hook. Rapid elongation and root promotion are auxin effects.
Option B: Leaf area expansion, chloroplast development (de-etiolation/skotomorphogenesis to photomorphogenesis), and flowering are responses to light (phytochrome, cryptochrome, photoperiodism), not ethylene.
Option D: Fruit ripening and leaf abscission are indeed ethylene effects, but phototropic bending is mediated by auxin (lateral redistribution of IAA in response to unilateral light), not ethylene.

Answer: (C) ← Go back to Q27

Q28.

Solution

Concept — Active Ion Uptake in Root Hairs: Proton Motive Force and H^+-ATPase

Plants do not have a Na^+/K^+-ATPase (an animal adaptation). Instead, the primary active transport pump in plant plasma membranes is the H^+-ATPase (proton pump), which hydrolyses ATP to export H^+ out of the cell. This creates two components of a proton motive force: (i) a chemical gradient (ΔpH, inside more alkaline) and (ii) an electrical potential ($\Delta\Psi$, inside negative ≈ -120 to -170 mV). Cations (K^+, Ca^{2+}, NH_4^+) enter passively down the electrochemical gradient via specific channel proteins; anions (NO_3^-, $H_2PO_4^-$, SO_4^{2-}) are taken up via H^+-coupled symporters that use the proton gradient as the driving force.

Step 1 — K^+ uptake:

At low external K^+ concentrations (typical of most soils), K^+ is taken up via high-affinity K^+ transporters (e.g., AtHAK5 in Arabidopsis), which are H^+/K^+ symporters driven by the proton gradient. At higher external K^+, inward-rectifying K^+ channels (e.g., AKT1) suffice.

Step 2 — Role of H^+-ATPase in nutrient acquisition:

Phosphate uptake: H^+/Pi co-transporters on mycorrhizal fungi and plant root cells are energised by the proton gradient. Nitrate uptake: NRT1/NRT2 family transporters are H^+-coupled. Soil acidification by H^+ extrusion also promotes dissolution of soil mineral nutrients.

Why other options are wrong:

Option A: Na^+/K^+-ATPase is an animal-specific pump; plants do not possess this enzyme. The analogous primary pump in plants is H^+-ATPase.

Option B: Osmosis moves water in response to water potential gradients; it does not carry dissolved solutes (solute drag exists but is negligible for most mineral ions at physiological flow rates). Ion uptake in plants is driven by electrochemical gradients, not osmotic flow.

Option C: ATP is not physically attached to transport proteins in the way described; rather, ATP is hydrolysed by the H^+-ATPase pump, and the resulting proton gradient then drives secondary active transporters. The ions are not physically "carried" with ATP.

Answer: (D) ← Go back to Q28

Q29.

Solution

Concept — Fat Mobilisation in Germinating Seeds: Glyoxysomes and Glyoxylate Cycle

Oilseeds (castor bean, sunflower, peanut, soybean) store most of their energy reserves as triacylglycerols (TAGs) in oil bodies (oleosomes). During germination, before the seedling can photosynthesise, the stored fat must be converted to sucrose (the major transport sugar) to fuel growth of the embryonic axis. The pathway: TAG $\xrightarrow{\text{lipase}}$ fatty acids + glycerol $\xrightarrow{\text{glyoxysome (beta-oxidation)}}$ acetyl-CoA $\xrightarrow{\text{glyoxylate cycle}}$ succinate $\xrightarrow{\text{mitochondria (TCA + gluconeogenesis)}}$ sucrose.

Step 1 — Glyoxysomes:

Glyoxysomes are specialised peroxisomes that contain: (a) the beta-oxidation enzyme set (acyl-CoA oxidase, MFP, thiolase) and (b) the two unique glyoxylate cycle enzymes: isocitrate lyase and malate synthase. These enzymes allow the net conversion of 2 acetyl-CoA to 1 succinate (bypassing the two CO_2-releasing decarboxylation steps of the TCA cycle). Succinate is then exported to mitochondria.

Step 2 — Conversion to sucrose:

In mitochondria, succinate $\rightarrow$ oxaloacetate (via TCA). Oxaloacetate is exported to the cytosol $\rightarrow$ phosphoenolpyruvate carboxykinase (PEPCK) $\rightarrow$ PEP $\rightarrow$ gluconeogenesis $\rightarrow$ fructose-6-phosphate $\rightarrow$ sucrose-6-phosphate synthase $\rightarrow$ sucrose $\rightarrow$ transported to seedling axis.

Why other options are wrong:

Option B: Chloroplasts do not have the enzymatic machinery to oxidise fatty acids, nor do vacuoles. Light-driven fatty acid to carbohydrate conversion does not occur; fatty acid oxidation is a purely catabolic process.

Option C: The nucleus contains genetic information (DNA) but no metabolic enzymes for fatty acid oxidation; the rough ER synthesises proteins but does not convert fatty acids to glucose.

Option D: The Golgi processes and packages secretory proteins and polysaccharides; the smooth ER is involved in lipid synthesis and detoxification, not fatty acid oxidation for gluconeogenesis.

Answer: (A) ← Go back to Q29

Q30.

Solution

Concept — Calvin Cycle: CO_2 Fixation by RuBisCO

The Calvin cycle (C3 cycle, photosynthetic carbon reduction cycle) proceeds in three stages: (1) Carboxylation: RuBisCO catalyses the addition of CO_2 to ribulose-1,5-bisphosphate (RuBP, a 5-carbon molecule) to form an unstable 6-carbon intermediate that immediately splits into two molecules of 3-

phosphoglycerate (3-PGA, a 3-carbon compound). (2) Reduction: 3-PGA is phosphorylated by ATP (via PGK) and then reduced by NADPH (via GAPDH) to give glyceraldehyde-3-phosphate (G3P). (3) Regeneration: G3P is used to regenerate RuBP using ATP (via PRK), allowing continued CO_2 fixation.

Step 1 — RuBisCO as the world's most abundant enzyme:
RuBisCO (ribulose-1,5-bisphosphate carboxylase/oxygenase) constitutes up to 50% of soluble leaf protein. Its carboxylase activity fixes CO_2; its oxygenase activity initiates photorespiration (adds O_2 to RuBP instead of CO_2). The slow catalytic rate and dual specificity are among the reasons why plants invest so heavily in RuBisCO protein.

Step 2 — Net output:
For every 3 CO_2 fixed: 3 ATP + 2 NADPH consumed per CO_2 fixed (total: 9 ATP + 6 NADPH per G3P net output). One G3P is the net carbohydrate gain; five G3P are used to regenerate three RuBP.

Why other options are wrong:
Option A: Phosphoglycerate kinase (PGK) catalyses the phosphorylation of 3-PGA to 1,3-bisphosphoglycerate in the *reduction* step (not carboxylation); it does not incorporate CO_2.
Option C: Ferredoxin-$NADP^+$ reductase (FNR) reduces $NADP^+$ to NADPH in the light reactions (electron transport chain at PSI); it is not part of the Calvin cycle and does not fix CO_2.
Option D: Phosphoribulokinase (PRK) phosphorylates ribulose-5-phosphate to regenerate RuBP in the regeneration stage; it does not fix CO_2 and acts after, not at, the point of CO_2 incorporation.

Answer: (B) ← Go back to Q30

Q31.

Solution

Concept — Copper-T IUD: Mechanism of Contraceptive Action
The copper intrauterine device (IUD; e.g., Copper-T 380A) is a highly effective (>99%), long-acting, reversible contraceptive. It consists of a T-shaped polyethylene frame wound with copper wire (≈380 mm^2 copper surface area), placed in the uterine cavity by a clinician. Copper ions (Cu^{2+}) are continuously released at low concentrations into the uterine fluid and cervical mucus.

Step 1 — Spermicidal mechanism:
Cu^{2+} ions are directly toxic to spermatozoa: they impair sperm motility (by inhibiting flagellar enzymes, particularly dynein ATPase), reduce sperm viability, and inhibit sperm capacitation. The physical presence of the IUD also alters the endometrial environment (inflammatory leukocyte recruitment) and alters cervi-

cal mucus, further reducing sperm penetration.

Step 2 — Anti-implantation (secondary mechanism):
If fertilisation does occur (a rare event given the primary spermicidal effect), the copper-altered endometrial environment is less receptive to blastocyst implantation. The Copper-T can also function as emergency contraception (inserted within 5 days of unprotected intercourse, >99% effective).

Why other options are wrong:
Option A: The copper-T IUD does not physically block the cervix; it sits within the uterine cavity. The cervix remains patent; the copper-T's arm string hangs through the cervical os for removal.
Option B: The copper-T releases no hormones; it contains only copper. Hormone-releasing IUDs (e.g., Mirena, containing levonorgestrel/progestin) suppress ovulation and thicken cervical mucus, but the copper-T does not affect FSH, LH, or ovulation.
Option D: Sperm require a slightly alkaline cervical mucus environment for optimal motility; copper ions act by ionic toxicity on sperm, not by altering mucus pH to alkaline levels.

Answer: (C) ← Go back to Q31

Q32.

Solution

Concept — ZIFT (Zygote Intrafallopian Transfer) vs IVF Embryo Transfer
Assisted Reproductive Technologies (ARTs) include various procedures designed to achieve pregnancy when natural conception fails. In standard IVF (in vitro fertilisation), the embryo is typically transferred at the 4-to-8-cell stage (Day 2–3) or blastocyst stage (Day 5) into the uterine cavity via a transcervical catheter. ZIFT (Zygote Intrafallopian Transfer) is a variant in which the fertilised egg (zygote, Day 0–1, before cleavage begins or at the 1-cell stage) is transferred into the fallopian tube laparoscopically (requiring abdominal surgery).

Step 1 — Rationale for ZIFT:
The fallopian tube provides a natural physiological environment for early embryo development and transport. Advocates of ZIFT argued it may improve implantation compared to direct uterine transfer, but controlled studies have not consistently demonstrated superiority over conventional IVF with uterine transfer. ZIFT requires a patent fallopian tube (useless in tubal factor infertility) and an additional surgical procedure.

Step 2 — GIFT (Gamete Intrafallopian Transfer):
A closely related ART where unfertilised oocytes and prepared sperm are placed directly into the fallopian tube laparoscopically (fertilisation occurs in vivo, not in

vitro). GIFT is acceptable to some religious groups that object to in vitro fertilisation.

Why other options are wrong:
Option A: Transfer of a blastocyst (Day 5) into the uterus is standard IVF blastocyst transfer, *not* ZIFT. ZIFT uses the *zygote* stage.
Option B: Transfer through the cervix would be a uterine (not tubal) transfer; the morula stage is used in some IVF protocols (Day 3–4 uterine transfer), not ZIFT into the fallopian tube.
Option C: A 16-cell stage is a morula (Day 3–4); transferring it transcervically into the uterus is a form of IVF embryo transfer, not ZIFT (which requires laparoscopy + zygote).

Answer: (D) ← Go back to Q32

Q33.

Solution

Concept — Placental Endocrine Functions: Luteoplacental Shift
The placenta is unique in that it is a transient endocrine organ that takes over crucial hormonal functions from the corpus luteum during pregnancy. In the first 6–10 weeks of gestation, the corpus luteum (maintained by HCG from the trophoblast) produces progesterone and oestrogen to maintain the decidualised endometrium. Around weeks 10–12, the luteoplacental shift occurs: the placenta becomes the dominant source of progesterone (from cholesterol, via the syncytiotrophoblast mitochondria and smooth ER) and oestrogens (requiring fetal adrenal DHEA-S as a precursor). The corpus luteum then regresses.

Step 1 — Hormones produced by the placenta:
(i) HCG (human chorionic gonadotropin): peaks at 8–10 weeks, maintains corpus luteum; (ii) Progesterone: maintains uterine quiescence (prevents premature labour), promotes endometrial secretions; (iii) Oestrogens (oestradiol, oestriol): promote uterine growth, stimulate breast development; (iv) hPL (human placental lactogen / chorionic somatomammotropin): promotes maternal lipolysis (sparing glucose for fetus), mammary gland growth; (v) CRH (corticotropin-releasing hormone from placenta): implicated in timing of parturition.

Step 2 — Clinical relevance:
In ectopic pregnancy (tubal), the trophoblast secretes HCG, but the placenta cannot develop fully; HCG levels rise abnormally slowly. Detection of urinary/plasma HCG is the basis of pregnancy tests.

Why other options are wrong:
Option B: The placenta does not produce FSH or LH; these are anterior pituitary gonadotrophins. FSH and LH are suppressed during pregnancy by high proges-

terone and oestrogen (negative feedback), preventing ovulation.
Option C: The placenta does not produce glucagon; blood glucose regulation involves maternal pancreatic insulin and glucagon, and the fetus itself produces insulin by mid-gestation.
Option D: The placenta does not produce renin; renin is produced by the juxtaglomerular apparatus of the kidney. Pre-eclampsia involves placental and endothelial dysfunction, but is not due to placental renin secretion.

Answer: (A) ← Go back to Q33

Q34.

Solution

Concept — Allopatric Speciation: Role of Geographic Isolation
Speciation is the process by which a single ancestral population gives rise to two or more reproductively isolated descendant species. In allopatric speciation (Greek: "other country"), the critical first step is physical separation of populations by a geographical barrier (vicariance): mountain range, ocean, river, desert, or glacier. Once isolated, the two populations cannot exchange genes; they independently accumulate mutations, adapt to different local environments via natural selection, and may experience different genetic drift. Over time (thousands to millions of years), pre- and post-zygotic reproductive isolating mechanisms (different mating seasons, courtship behaviours, gamete incompatibilities, hybrid inviability, sterility) accumulate until interbreeding is no longer possible even if the barrier disappears — speciation is complete.

Step 1 — Evidence:
Darwin's finches on Galápagos islands: a single mainland finch species colonised the islands; geographic isolation on different islands led to 14+ species with diverse beak morphologies. African cichlid fishes in isolated rift lakes: hundreds of species from a few ancestors due to lake isolation.

Step 2 — Allopatric vs sympatric:
Sympatric speciation (same geographic area): occurs most clearly in plants via polyploidy (instantaneous reproductive isolation), or in phytophagous insects shifting to new host plants. In animals, sympatric speciation is rarer and more controversial (e.g., cichlids within a single lake via sexual selection).

Why other options are wrong:
Option A: A single mutant individual reproductively isolated within a population describes a potential step toward sympatric speciation (or the initiation of a new lineage) but is not the mechanism of allopatric speciation, which requires *geographic* separation of a population (not just a single individual).
Option C: Large population size and resource competition describe conditions for

disruptive selection, which can contribute to sympatric speciation, not allopatric speciation.
Option D: Polyploidy causing instant reproductive isolation is the mechanism of sympatric speciation in plants (allopolyploidy) — it does not require geographic isolation and does not describe allopatric speciation.

Answer: (B) ← Go back to Q34

Q35.

Solution

Concept — Gause's Competitive Exclusion Principle (Law of Competitive Exclusion)
G.F. Gause (1934) cultured *Paramecium aurelia* and *P. caudatum* separately and together under the same conditions. When grown separately, both species exhibited logistic growth. When grown together competing for the same food (bacteria), *P. aurelia* always outcompeted and eliminated *P. caudatum* within ≈20 days. This led to the competitive exclusion principle: two species with identical ecological niches (same resources, same habitat) cannot coexist indefinitely; the superior competitor excludes the inferior one.

Step 1 — Coexistence mechanisms:
In nature, complete competitive exclusion is rare because most ecologically similar species have at least slightly different niches (resource partitioning). Character displacement (evolution of differences in resource use in response to competition — Darwin's finches beak sizes) allows coexistence. The "diversity-stability hypothesis" connects resource partitioning to community stability.

Step 2 — Lotka-Volterra model:
The competition equations ($dN_1/dt = r_1N_1(K_1 - N_1 - \alpha_{12}N_2)/K_1$) predict outcomes of competition: coexistence if intraspecific competition exceeds interspecific competition; exclusion otherwise.

Why other options are wrong:
Option A: Predation reducing species dominance is the Paine "keystone predator" concept (e.g., starfish maintaining mussel bed diversity), which is separate from Gause's competitive exclusion principle about interspecific competition.
Option B: Evolution toward similarity through competition is character convergence; the opposite (character displacement, divergence) is far more commonly documented as a consequence of competition.
Option D: Competitive exclusion does not always lead to extinction within one generation; it is typically a slow process occurring over many generations. Also, the winner does not necessarily undergo "rapid expansion" beyond its carrying capacity.

Answer: (C) ← Go back to Q35

Q36.

Solution

Concept — Decomposition: Stages and Humification

Decomposition of dead organic matter (detritus) is an essential ecosystem process that returns nutrients from biomass to the inorganic pool. The major sequential steps: (1) **Fragmentation**: physical breakdown of litter by detritivores (earthworms, woodlice, millipedes) into smaller pieces, increasing surface area for microbial attack. (2) **Catabolism/Leaching**: enzymes (cellulases, ligninases, proteases) secreted by bacteria and fungi hydrolyse polymers; water-soluble products leach into soil water. (3) **Humification**: partial breakdown products (modified lignin polymers, polyphenols, microbial cell wall components, melanins) condense into complex, dark-coloured, recalcitrant organic colloids called **humus**. Humus is very stable (residence time of years to centuries), adsorbs cations (high CEC), and improves soil structure. (4) **Mineralisation**: humus slowly decomposed by specialised microbes, releasing inorganic ions (NH_4^+, NO_3^-, PO_4^{3-}, K^+, Ca^{2+}).

Step 1 — Properties of humus:

Humus: dark brown/black colour; large surface area; negatively charged (due to carboxyl, phenolic groups) adsorbing Ca^{2+}, Mg^{2+}, K^+; improves soil aggregate stability, aeration, and water-holding capacity; provides slow-release nutrients; cation exchange capacity (CEC). Humus is the basis of soil fertility.

Step 2 — Rate of humification:

Humification rate is reduced in cold, dry, or acidic conditions (e.g., peat bogs accumulate partially decomposed material — Sphagnum moss peat). Warm, moist, slightly alkaline soils promote rapid humification and subsequent mineralisation.

Why other options are wrong:

Option A: Physical fragmentation of leaf litter by detritivores is the first stage of decomposition (fragmentation), not humification. Humification occurs *after* initial fragmentation and catabolism.

Option B: Leaching of water-soluble nutrients into soil water is the leaching subprocess; it transfers soluble compounds (simple sugars, amino acids, nitrates) but is distinct from humus formation.

Option C: Complete mineralisation (release of inorganic ions from organic matter) is the final step of decomposition; it is distinct from humification, which produces *stable* recalcitrant humus rather than fully mineralised ions.

Answer: (D) ← Go back to Q36

Q37.

Solution

Concept — Desert Biome: Xerophytic and Faunal Adaptations

The desert biome is characterised by low annual rainfall (<250 mm), extreme diurnal temperature fluctuations, high solar radiation, and low humidity. Organisms in this biome have evolved remarkable morphological, physiological, and behavioural adaptations to survive water scarcity and heat stress.

Step 1 — Plant adaptations (xerophytes):

CAM metabolism (Crassulacean Acid Metabolism): stomata open at night to fix CO_2 into malic acid (stored in vacuoles); stomata close during the day (avoiding water loss); malate decarboxylated during the day to supply CO_2 to RuBisCO. Succulents (cacti, aloe): store water in fleshy stems or leaves (parenchyma cells with large vacuoles). Spines (modified leaves or stipules): minimise transpiration surface area, reduce herbivory, and create a boundary layer reducing convective heat gain. Extensive shallow root systems (some exceeding 15 m lateral spread) for rapid uptake of brief rainfall.

Step 2 — Animal adaptations:

Nocturnal behaviour: avoids peak daytime heat and desiccation. Concentrated urine (uricotelic in reptiles, near-isotonic in some rodents): minimises urinary water loss. Aestivation (summer dormancy): desert snails, lungfish, spadefoot toads enter a dormant state during the hot, dry season. Burrowing: kangaroo rats, fennec foxes, gerbils use cool underground microhabitats.

Why other options are wrong:

Option B: Broad thin leaves for maximum light capture and thick fur for cold insulation are adaptations of shaded forest understorey plants and Arctic/Alpine mammals, respectively — not desert adaptations.

Option C: Buttress roots and epiphytic habits are tropical rainforest adaptations (to waterlogged soils and canopy competition for light). Arboreal locomotion and frugivory describe tropical rainforest animals.

Option D: Submerged leaves with aerenchyma, webbed feet, and gill slits in larvae describe aquatic/semi-aquatic organisms in freshwater or wetland biomes — the opposite extreme from desert.

Answer: (A) ← Go back to Q37

Q38.

Solution

Concept — Biological Oxygen Demand (BOD) as a Water Quality Indicator

BOD (Biological Oxygen Demand) is the amount of dissolved oxygen (DO) consumed by aerobic microorganisms while decomposing organic matter in a water sample at a given temperature (typically 20°C) over 5 days (BOD_5), measured in

mg O_2/L. It is an indirect measure of the organic matter (pollutant) concentration in water. Clean water: BOD <1–2 mg/L. Moderately polluted: BOD 2–8 mg/L. Heavily polluted (sewage effluent): BOD 200–400 mg/L.

Step 1 — Ecological consequence of high BOD:
When organic-rich sewage enters a river, heterotrophic bacteria multiply rapidly and consume dissolved O_2 at a rate exceeding its atmospheric replenishment (reaeration). DO falls, creating a hypoxic "oxygen sag curve" downstream of the discharge point. Fish and other aerobic aquatic organisms die or flee. Anaerobic decomposition then dominates, producing H_2S, CH_4, and NH_3 (foul odours). Recovery of DO occurs further downstream as organic matter is depleted.

Step 2 — BOD vs COD:
Chemical Oxygen Demand (COD) measures all chemically oxidisable material (including non-biodegradable compounds) using a strong oxidant ($K_2Cr_2O_7$). COD $\geq$ BOD always. For purely biodegradable organic waste (sewage): BOD/COD ratio is high ($\approx$0.5–0.8). Industrial effluents with non-biodegradable pollutants have a low BOD/COD ratio.

Why other options are wrong:
Option A: High BOD indicates low DO (due to microbial consumption), which is bad for aquatic life — the opposite of this option. High DO correlates with *low* BOD (clean water).
Option C: Photosynthetic activity by phytoplankton *produces* O_2 during the day, which would tend to *decrease* BOD (less O_2 needed by decomposers if organic loading is low). Algal blooms followed by die-off increase BOD, but the acute cause is organic pollution, not photosynthesis directly.
Option D: High BOD implies a *high* concentration of heterotrophic bacteria actively consuming oxygen during decomposition, not a low concentration.

Answer: (B) ← Go back to Q38

Q39.

Solution

Concept — CRISPR-Cas9: Role of the Guide RNA (gRNA)
CRISPR-Cas9 is a two-component system: (1) the Cas9 protein (a dual-lobe endonuclease with HNH and RuvC-like nuclease domains, each cleaving one strand of dsDNA) and (2) the guide RNA (gRNA). The gRNA is an engineered fusion of: (a) the CRISPR RNA (crRNA) containing a $\approx$20-nucleotide spacer sequence complementary to the genomic target and (b) the transactivating crRNA (tracrRNA) scaffold that binds Cas9. The gRNA directs the Cas9/gRNA ribonucleoprotein complex to a specific genomic locus by base-pairing of the spacer with the target strand ("protospacer"), provided an adjacent PAM sequence (5′-NGG-3′ for *S.*

pyogenes Cas9) is present on the non-target strand.

Step 1 — DNA cleavage:
After target recognition and R-loop formation, Cas9 undergoes conformational activation: the HNH domain cleaves the strand complementary to the gRNA; the RuvC domain cleaves the non-complementary strand — creating a blunt-ended double-strand break (DSB) 3 bp upstream of the PAM.

Step 2 — DSB repair and genome editing:
NHEJ (error-prone, fast): inserts/deletions (indels) at the cut site → frameshift → gene knockout. HDR (precise, requires a donor template with homologous arms): introduces specific sequence changes. Applications: disease modelling, gene therapy (e.g., sickle cell disease: correction of HBB mutation; Duchenne MD), agriculture (disease-resistant crops).

Why other options are wrong:
Option A: The gRNA alone has no nuclease activity; it is a non-coding RNA that acts purely as a targeting molecule. Cas9 protein provides all endonuclease activity. Neither component functions independently.
Option B: The gRNA is a non-coding RNA (ncRNA); it is not translated into protein and does not encode Cas9. Cas9 is expressed separately (from a plasmid or mRNA) and forms a complex with the pre-made gRNA.
Option D: The gRNA does not repair the DNA break; repair is carried out by the cell's own DNA repair machinery (NHEJ or HDR). If HDR is desired, a separate donor template (ssDNA oligo or plasmid with homology arms) must be supplied in addition to the Cas9/gRNA complex.

Answer: (C) ← Go back to Q39

Q40.

Solution

Concept — Stirred-Tank Bioreactor: Function of the Impeller
Industrial bioreactors are designed to maintain optimal conditions (temperature, pH, dissolved oxygen, nutrient concentration) for large-scale microbial or mammalian cell cultures producing biologics (insulin, monoclonal antibodies, vaccines, enzymes, recombinant proteins). The stirred-tank bioreactor (STR, also called a continuously stirred tank reactor, CSTR) is the most widely used configuration. Key components: vessel (stainless steel, glass), impeller (agitator), sparger (gas distributor), baffles (prevent vortex formation), probes (pH, DO, temperature, foam), inlet/outlet ports.

Step 1 — Impeller function:
The impeller (driven by a motor through a top- or bottom-entry shaft) rotates to agitate the culture broth. Agitation serves multiple critical purposes: (i) *Mixing*:

ensures homogeneous distribution of nutrients (glucose, amino acids, salts), cells, and pH throughout the vessel (prevents concentration gradients). (ii) *O_2 mass transfer*: breaks up air/O_2 bubbles (from sparger) into smaller bubbles, greatly increasing gas-liquid interfacial area and enhancing O_2 transfer into the liquid phase (kLa). (iii) *CO_2 stripping*: removes metabolic CO_2 from the broth. (iv) *Heat distribution*: homogenises temperature.

Step 2 — Sparger function (distinct from impeller):
The sparger (porous ring or pipe at the bioreactor base) distributes fine bubbles of sterile air/O_2/N_2 into the broth. The impeller then further disperses these bubbles. Baffles (vertical plates on the vessel wall) disrupt rotational flow and increase turbulence.

Why other options are wrong:
Option A: Air filtration and sterilisation are performed *before* the sparger, by inline HEPA or membrane filters on the gas inlet line. The impeller does not filter air.
Option B: Bioreactor sterility is maintained by autoclave sterilisation (vessel and medium), in-line steam sterilisation of connections, positive pressure (slightly above atmospheric) inside the vessel (preventing ingress of contaminants), and sterile filtered gas — not by the impeller creating a "positive pressure zone at the top."
Option C: Product harvesting is a separate downstream process: either continuous (perfusion bioreactors with cell retention filters) or batch (centrifugation/filtration at end of run). The impeller does not harvest product.

Answer: (D) ← Go back to Q**40**

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	2	B	3	C	4	D	5	A
6	B	7	C	8	D	9	A	10	B
11	C	12	D	13	A	14	B	15	C
16	D	17	A	18	B	19	C	20	D
21	A	22	B	23	C	24	D	25	A
26	B	27	C	28	D	29	A	30	B
31	C	32	D	33	A	34	B	35	C
36	D	37	A	38	B	39	C	40	D