GUJCET Biology

Sample Paper – 8

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 gastric glands of the stomach contain several specialised cell types, each with distinct secretory roles. Which cell type secretes hydrochloric acid (HCl) into the stomach lumen, and what is the membrane-pump mechanism responsible?

A. Parietal (oxyntic) cells, via the H^+/K^+-ATPase (proton pump) on their apical membrane

B. Chief (peptic) cells, which secrete pepsinogen; HCl is a metabolic by-product of pepsinogen activation

C. G cells (enteroendocrine cells), which secrete gastrin that stimulates HCl production in neighbouring cells

D. Mucous neck cells, which secrete bicarbonate-rich mucus to neutralise luminal HCl

Q2. A 22-year-old male develops bloating, flatulence, and watery diarrhoea within two hours of consuming milk or dairy products. Investigations reveal a deficiency of a specific brush-border enzyme in the small intestinal epithelium. Which enzyme is deficient, and where in the gut is it normally expressed?

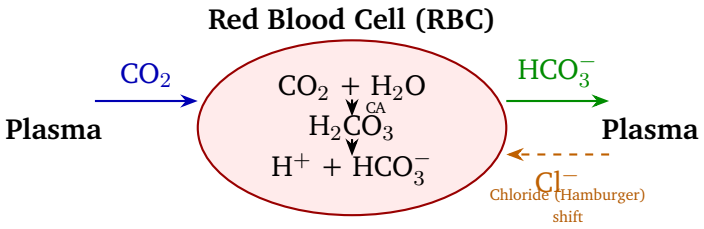
A. Sucrase, found in the pancreatic juice secreted into the duodenal lumen

B. Lactase (lactase-phlorizin hydrolase), located at the brush border (microvilli) of small intestinal enterocytes

C. Salivary amylase, produced by the parotid glands and responsible for carbohydrate digestion in the mouth

D. Pepsin, secreted as pepsinogen by chief cells in the gastric mucosa

Q3. Carbon dioxide (CO_2) produced by tissues is transported in blood via three mechanisms. The figure below illustrates CO_2 handling inside a red blood cell (RBC) and the associated ion exchange with the plasma.



What percentage of CO_2 is transported as bicarbonate ions (HCO_3^-) in the plasma, and what ion exchange mechanism maintains electrical neutrality in the RBC?

A. Approximately 7–10%, by simple dissolution of CO_2 directly in plasma

B. Approximately 20–25%, as carbamino-haemoglobin formed by CO_2 binding to the globin amino groups of Hb

C. Approximately 70%, as HCO_3^- ions; electrical neutrality is maintained by the chloride (Hamburger) shift in which HCO_3^- exits the RBC and Cl^- enters

D. Approximately 50%, by CO_2 binding directly to the haem (iron-containing) group of haemoglobin

Q4. Respiratory quotient (RQ) is defined as the ratio of CO_2 produced to O_2 consumed during metabolism of a substrate. A person is placed on a strict carbohydrate-free diet and metabolises *only* fat as an energy source. What would be the approximate RQ of this person?

A. 1.0

B. 0.8

C. 1.2

D. 0.7

Q5. An Rh-negative mother delivers her first Rh-positive baby without incident. During this delivery, fetal Rh-positive RBCs enter the maternal circulation, and she subsequently develops anti-D (Rh) IgG antibodies. During her second Rh-positive pregnancy, these antibodies cross the placenta and destroy fetal RBCs. Which condition does this describe?

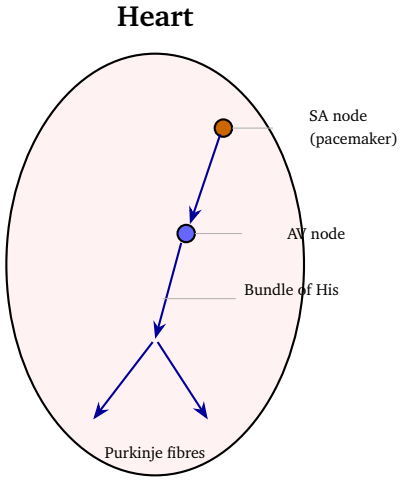
A. Erythroblastosis fetalis (haemolytic disease of the newborn)

B. Thalassaemia

C. Polycythaemia vera

D. Sickle-cell crisis

Q6. The heart has an intrinsic conduction system that coordinates its contractions. The figure below shows the pathway of electrical impulse conduction from the sinoatrial (SA) node through the heart.



Why is the sinoatrial (SA) node called the 'pacemaker' of the heart?

A. It is the largest and most muscular node in the heart's conduction system

B. It spontaneously generates electrical impulses at the highest intrinsic rate (≈70–75 bpm) and thus sets the rhythm for the entire heart

C. It directly receives neural signals from the cardiac centre of the medulla oblongata without any intermediary structure

D. It is located at the junction of the interventricular septum and the ventricular free wall

Q7. After digestion and absorption in the small intestine, nutrients are transported to the liver for first-pass metabolism before entering the systemic circulation. Which blood vessel specifically carries absorbed nutrients (glucose, amino acids, short-chain fatty acids) from the small intestine directly to the liver?

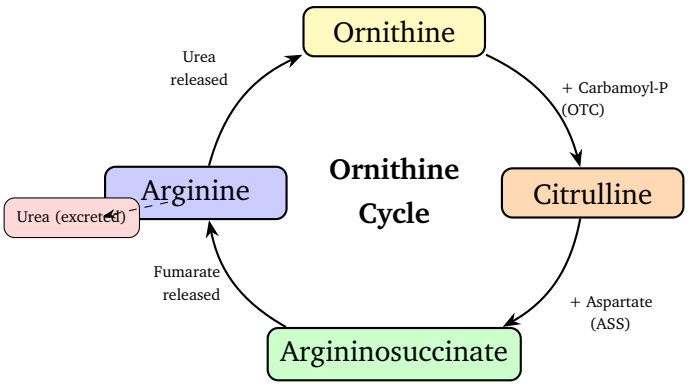
A. Hepatic artery

B. Hepatic vein

C. Hepatic portal vein

D. Superior mesenteric artery

Q8. The ornithine (urea) cycle converts toxic ammonia to water-soluble urea in the liver. The cycle is illustrated below.



Which enzyme catalyses the first committed step of the ornithine cycle — the condensation of ornithine with carbamoyl phosphate to form citrulline?

A. Arginase

B. Argininosuccinate lyase (ASL)

C. Carbamoyl phosphate synthetase I (CPS-I)

D. Ornithine transcarbamylase (OTC)

Q9. Animals are classified by their primary nitrogenous excretory product: ammonotelic (ammonia), ureotelic (urea), or uricotelic (uric acid). Which of the following organisms is CORRECTLY classified as uricotelic?

A. Pigeon (*Columba livia*)

B. Adult frog (*Rana tigrina*)

C. Salmon (*Salmo salar*)

D. Earthworm (*Pheretima posthuma*)

Q10. The glomerular filtration barrier consists of three layers: the fenestrated capillary endothelium, the glomerular basement membrane (GBM), and podocytes with slit diaphragms. Why are large plasma proteins such as albumin ($M_r \approx 69{,}000$ Da) absent from normal glomerular filtrate, even though they are present in blood?

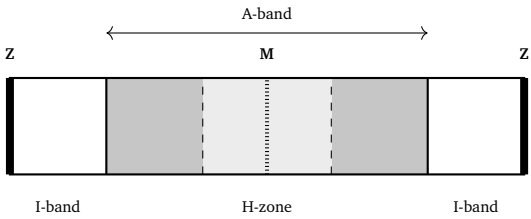
A. Plasma proteins are actively secreted back from the tubular lumen into the peritubular capillaries by proximal tubular cells

B. The filtration barrier (GBM and podocyte slit diaphragms) excludes them by molecular size and their net negative charge, which repels the negatively charged GBM proteoglycans

C. Plasma proteins are too few in number in blood to appear in the filtrate at detectable concentrations

D. Plasma proteins bind tightly to glucose, which is completely reabsorbed in the proximal convoluted tubule

Q11. The figure below represents a sarcomere — the functional contractile unit of a myofibril, bounded by two Z-lines.



During muscle contraction (as the sarcomere shortens), which bands or zones DECREASE in width?

A. A-band only

B. A-band and I-band

C. I-band and H-zone

D. H-zone only

Q12. In skeletal muscle, contraction is initiated when Ca^{2+} is released from the sarcoplasmic reticulum into the cytoplasm. What is the specific molecular role of Ca^{2+} in triggering cross-bridge formation between actin and myosin?

A. Ca^{2+} provides the chemical energy required for the power stroke by acting as an electron donor to myosin ATPase

B. Ca^{2+} directly hydrolyses ATP to ADP + P_i, releasing the energy required to cock the myosin head

C. Ca^{2+} stimulates the motor nerve terminal to release more acetylcholine, amplifying the neuromuscular signal

D. Ca^{2+} binds to troponin C, causing a conformational change that shifts tropomyosin away from the actin binding sites, exposing them for myosin head attachment

Q13. Different brain regions have distinct and specialised functions. A patient suffers a stroke confined to the cerebellum. Which functional deficit would MOST likely be observed?

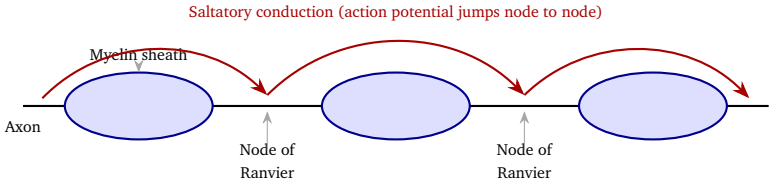
A. Loss of coordination and balance (ataxia), inability to perform rapid alternating movements (dysdiadochokinesia), and intention tremor

B. Loss of all conscious somatic sensation from the contralateral side of the body

C. Inability to form new explicit (declarative) memories (anterograde amnesia)

D. Loss of voluntary control of the respiratory rhythm

Q14. The figure below shows a segment of a myelinated nerve fibre. Nodes of Ranvier are visible as gaps between the myelin sheaths.



Why does saltatory conduction in myelinated nerve fibres result in faster impulse transmission compared to continuous conduction in unmyelinated fibres?

A. Myelin sheaths contain additional voltage-gated Na^+ channels that amplify the action potential at each internodal segment

B. The action potential is regenerated only at the unmyelinated nodes of Ranvier, effectively 'jumping' from node to node and covering greater distance per unit time

C. Myelinated fibres have a smaller axonal diameter, which reduces internal resistance and allows faster ion flow

D. The myelin sheath secretes neurotransmitters at each node that chemically relay the signal faster than electrical conduction

Q15. Insulin is the primary anabolic hormone that lowers blood glucose. After binding to its tyrosine kinase receptor on target cells, insulin triggers a signalling cascade. Through which specific mechanism does insulin promote glucose uptake in skeletal muscle and adipose tissue?

A. Insulin directly forms a glucose channel in the plasma membrane, creating a pore through which glucose enters the cell down its concentration gradient

B. Insulin activates glycogen phosphorylase, causing intracellular glycogen breakdown that creates a sink for incoming glucose

C. Insulin causes the translocation of GLUT-4 (glucose transporter type 4) vesicles from the cytoplasm to the plasma membrane, increasing glucose uptake capacity

D. Insulin activates glucokinase (hexokinase IV) in skeletal muscle to phosphorylate glucose and trap it intracellularly

Q16. Parathyroid hormone (PTH) is a key regulator of calcium and phosphate homeostasis. A patient is diagnosed with hypoparathyroidism (inadequate PTH secretion). Which set of clinical and biochemical findings would be EXPECTED?

A. High blood Ca^{2+}, low serum phosphate, and increased osteoclast activity

B. High blood Ca^{2+}, increased bone resorption, and reduced urinary Ca^{2+} excretion

C. Normal serum Ca^{2+} with compensatory increased urinary Ca^{2+} loss

D. Low blood Ca^{2+} (hypocalcaemia) causing neuronal hyperexcitability, muscle tetany, and seizures

Q17. Ribosomes are the sites of protein synthesis in all living cells and differ in size between prokaryotes and eukaryotes — a distinction exploited by many antibiotics. Which combination CORRECTLY describes ribosomal subunit composition?

A. Prokaryotic ribosomes: 70S (50S large + 30S small subunit); cytoplasmic eukaryotic ribosomes: 80S (60S large + 40S small)

B. Prokaryotic ribosomes: 80S (60S + 40S); cytoplasmic eukaryotic ribosomes: 70S (50S + 30S)

C. Prokaryotic ribosomes: 70S (60S large + 40S small); eukaryotic ribosomes: 80S (50S + 30S)

D. Both prokaryotic and eukaryotic cytoplasmic ribosomes are 80S, differing only in their protein composition

Q18. The endoplasmic reticulum (ER) is classified as rough ER (rER, bearing ribosomes) or smooth ER (sER, lacking ribosomes). A liver hepatocyte is actively synthesising steroid hormones and metabolising drugs via its cytochrome P450 enzyme system. Which organelle would be present in GREATEST abundance in this cell?

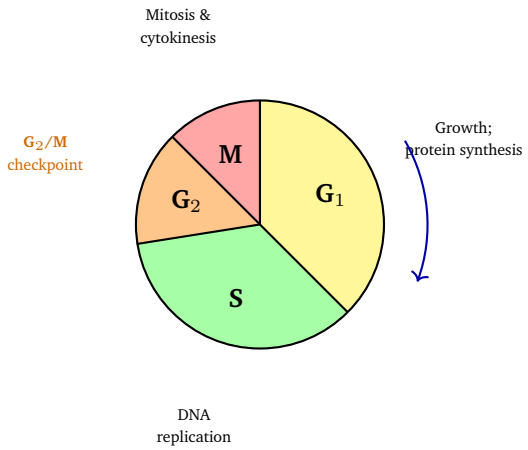
A. Rough ER, because steroid synthesis is initiated on membrane-bound ribosomes

B. Smooth ER, because it houses the enzymes for lipid and steroid biosynthesis and contains the drug-detoxifying cytochrome P450 system

C. Rough ER, because drug detoxification requires the secretion of large quantities of proteolytic enzymes into the blood

D. Both rough and smooth ER in equal proportions, as steroid and secretory-protein synthesis are always tightly co-regulated

Q19. The cell cycle is a series of tightly regulated phases. The figure below illustrates the four major phases and the relative timing of key checkpoints.



At which cell cycle checkpoint is the cell specifically monitored to ensure that DNA replication has been completed accurately before it is allowed to enter mitosis?

A. G1/S checkpoint (restriction point) — monitors cell size and nutrient availability, and DNA integrity before replication begins

B. Spindle assembly checkpoint (SAC) — monitors for correct kinetochore–microtubule attachments during mitosis

C. G2/M checkpoint — monitors for complete and accurate DNA replication before the cell proceeds to mitosis

D. G1 DNA-damage checkpoint — detects irreparable DNA damage and triggers apoptosis, distinct from the G2/M checkpoint

Q20. In 1902, Walter Sutton (studying grasshopper chromosomes) and Theodor Boveri (studying sea urchin embryos) independently proposed the chromosomal theory of inheritance. What was the primary significance of this theory?

A. It proved that DNA, not protein, is the genetic material by demonstrating specific X-ray diffraction patterns of chromosomes

B. It established that genes are made of protein and that chromosomes are merely structural scaffolding for protein

C. It explained how mutations arise as specific chemical changes in a DNA sequence

D. It provided the physical and cytological basis for Mendel's laws by demonstrating that chromosomes segregate and assort independently during meiosis, mirroring the behaviour of Mendel's hereditary factors

Q21. A plant displaying the dominant purple-flower phenotype (gene F: purple, dominant over f: white) is crossed with a white-flowered plant (ff). The offspring consist of 50% purple-flowered plants and 50% white-flowered plants. What is the genotype of the purple-flowered parent?

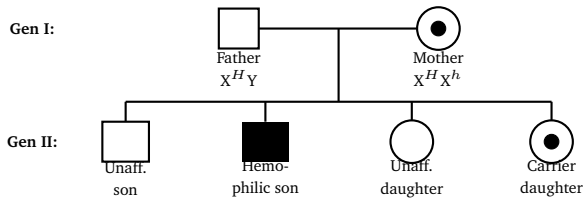
A. Ff (heterozygous dominant)

B. FF (homozygous dominant)

C. ff (homozygous recessive) — impossible since the parent shows purple phenotype

D. The genotype cannot be determined from a test cross; additional selfing experiments are needed

Q22. Haemophilia A is an X-linked recessive disorder caused by deficiency of clotting factor VIII. A carrier mother (X^HX^h) and an unaffected father (X^HY) have children. The pedigree below represents this family.



What is the probability that a SON born to this couple will be haemophilic?

A. 25%

B. 50%

C. 75%

D. 0% (sons of unaffected fathers cannot be haemophilic)

Q23. Down syndrome is the most common autosomal chromosomal disorder in humans, characterised by intellectual disability, characteristic facial features, and 47 total chromosomes. What is the chromosomal basis of the most common form of Down syndrome?

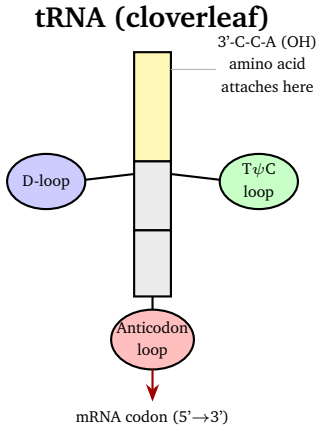
A. Monosomy of chromosome 21 (only one copy of chr 21), resulting in 45 chromosomes total

B. Trisomy of chromosome 18 (three copies of chr 18), which also produces intellectual disability and a total of 47 chromosomes

C. Trisomy 21 — three copies of chromosome 21 due to non-disjunction during meiosis in a parent, giving 47 chromosomes

D. Deletion of the long arm (q arm) of chromosome 21, causing intellectual disability without the classic Down syndrome karyotype

Q24. Transfer RNA (tRNA) adopts a characteristic cloverleaf secondary structure with several functionally distinct regions. The figure below shows a schematic of a tRNA molecule.



Which structural region of tRNA base-pairs with the mRNA codon during translation at the ribosome?

A. Acceptor stem (3'-CCA-OH end), where the activated amino acid is covalently attached

B. D-loop (dihydrouridine loop), which is recognized by aminoacyl-tRNA synthetase enzymes

C. TψC loop (TpsiC loop), which interacts with the ribosome and elongation factors

D. Anticodon loop, which contains three nucleotides complementary to the mRNA codon

Q25. The *trp* operon of *E. coli* controls the biosynthesis of the amino acid tryptophan. Unlike the inducible *lac* operon, the *trp* operon is a repressible system. When intracellular tryptophan levels are HIGH, what occurs?

A. Tryptophan acts as a corepressor, binding to the inactive trp repressor protein to form an active repressor–corepressor complex that binds the operator and blocks transcription of the trp biosynthesis genes

B. Tryptophan directly binds to the operator DNA and physically prevents RNA polymerase from passing without involving the repressor protein

C. High tryptophan induces transcription of the trp genes via positive feedback, producing more biosynthetic enzymes

D. Tryptophan binds to and inactivates the trp repressor, preventing it from binding the operator and thus allowing continued transcription

Q26. In eukaryotic cells, the primary transcript of a protein-coding gene (pre-mRNA or hnRNA) undergoes several processing steps before export to the cytoplasm. Which statement CORRECTLY describes RNA splicing?

A. Introns (intervening sequences) are exported to the cytoplasm where they are translated into regulatory proteins and then degraded

B. Introns are removed from the pre-mRNA by the spliceosome (a complex of snRNPs and associated proteins), and the exons are ligated to form a mature, translatable mRNA

C. RNA splicing occurs at the ribosome simultaneously with translation, coupling transcription and translation as in prokaryotes

D. Introns are removed by restriction endonucleases in the nucleus before export of the mature mRNA to the cytoplasm

Q27. Photoperiodism is the ability of plants to measure the duration of light and darkness in a 24-hour cycle to regulate seasonal flowering. Which statement about photoperiodism is CORRECT?

A. Short-day plants (SDPs) require the light period to EXCEED a critical minimum duration in order to initiate flowering

B. Long-day plants (LDPs) require the night period to EXCEED a critical maximum duration in order to flower

C. Short-day plants actually require a sufficiently long, uninterrupted dark period (critical night length) to flower; even a brief interruption of the dark period with red light prevents flowering in SDPs

D. Day-neutral plants (e.g. tomato) require exactly equal durations of light and dark (12 h:12 h) to initiate flowering

Q28. Abscisic acid (ABA) is often called the 'stress hormone' in plants. During drought (water stress), ABA levels rise sharply. Through which specific

mechanism does ABA trigger stomatal closure to reduce transpirational water loss?

A. ABA stimulates K^+ influx into guard cells, causing them to swell by osmosis and thereby close the stomatal pore

B. ABA activates aquaporins that actively pump water into guard cells, increasing their turgor pressure to mechanically seal the pore

C. ABA directly blocks the stomatal pore by inducing rapid callose deposition across the aperture

D. ABA triggers the efflux of K^+ (and Cl^-) from guard cells; the resulting osmotic loss of water reduces guard cell turgor pressure, causing stomata to close

Q29. CAM (Crassulacean Acid Metabolism) plants are highly adapted for arid environments. In CAM plants, stomata open at night rather than during the day. Which statement CORRECTLY explains the adaptive advantage and mechanism of CAM photosynthesis?

A. At night, CO_2 enters through open stomata and is fixed by PEP carboxylase into oxaloacetate, then converted to malate, which is stored in vacuoles. During the day, stomata remain closed (conserving water), stored malate is decarboxylated to release CO_2, which feeds the Calvin cycle

B. CAM plants open stomata at night because lower temperatures allow RuBisCO to operate more efficiently and without photorespiration

C. The light reactions of photosynthesis occur at night (in the dark) in CAM plants, allowing CO_2 fixation by RuBisCO in the absence of photorespiration

D. CAM plants use the C4 pathway via spatially separated bundle sheath and mesophyll cells, identical in mechanism to C4 plants such as maize

Q30. In C3 plants, carbon dioxide fixation in the Calvin cycle (dark reactions) results in the immediate formation of the first stable organic compound.

What is this first stable product of CO_2 fixation, and how many carbon atoms does it contain?

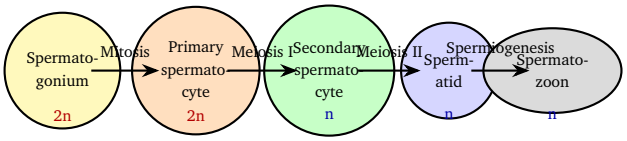
A. Oxaloacetate (OAA) — a 4-carbon dicarboxylic acid

B. 3-Phosphoglycerate (3-PGA) — a 3-carbon compound produced when RuBisCO adds CO_2 to ribulose-1,5-bisphosphate (RuBP)

C. Pyruvate — a 3-carbon α-keto acid formed during glycolysis

D. Phosphoenolpyruvate (PEP) — the primary 3-carbon CO_2 acceptor in C4 plants

Q31. Spermatogenesis is the process of sperm formation in the seminiferous tubules of the testis. The stages are shown in the figure below.



After meiosis II is completed and round spermatids are formed, what process converts them into mature spermatozoa?

A. Spermatids undergo a second round of mitotic cell division to produce a greater number of cells before differentiation

B. Spermatids undergo a second round of DNA replication before differentiating into motile spermatozoa

C. Spermiogenesis — a process of morphological differentiation (without further cell division) in which spermatids develop an acrosome-bearing head, mitochondria-rich midpiece, and a flagellar tail to form mature spermatozoa

D. Spermatids fuse with a nearby secondary oocyte to trigger the completion of their maturation process

Q32. Approximately 6–7 days after fertilisation, the blastocyst arrives in the uterus and undergoes implantation into the endometrium. Which cell layer of the blastocyst is primarily responsible for the invasive embedding into the uterine wall?

A. The inner cell mass (ICM / embryoblast) directly penetrates the endometrial epithelium and anchors the embryo

B. Progesterone released by the anterior pituitary stimulates the endometrium to actively engulf and internalise the blastocyst

C. The blastocyst simply adheres to the surface of the endometrial epithelium without any cellular invasion

D. The trophoblast (outer cell layer), which differentiates into the syncytiotrophoblast and cytotrophoblast, secretes proteolytic enzymes that digest the endometrial tissue and invasively embed the blastocyst

Q33. In flowering plants, normal reproduction involves fertilisation. However, some plants produce seeds or fruits without fertilisation. Which statement CORRECTLY defines and distinguishes apomixis and parthenocarpy?

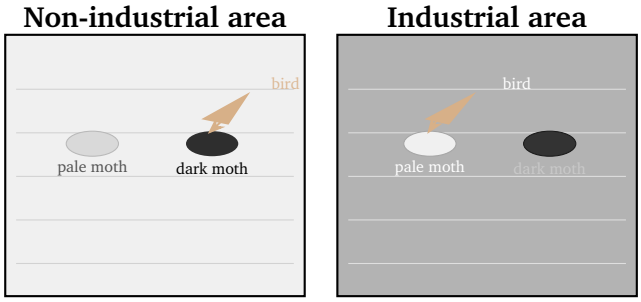
A. Apomixis is the formation of seeds without fertilisation (asexual seed production); parthenocarpy is the development of fruit without fertilisation — both bypass the normal double fertilisation of angiosperms

B. Apomixis requires pollination but not fertilisation, while parthenocarpy requires both pollination and fertilisation to produce seedless fruits

C. Both parthenocarpy and apomixis are triggered by double fertilisation, one producing only endosperm (seed) and the other producing only the pericarp (fruit)

D. Apomixis always produces diploid seeds via direct mitotic development; parthenocarpy always produces haploid fruits from unfertilised egg cells

Q34. The peppered moth (*Biston betularia*) in industrialised England showed a dramatic shift from predominantly pale to predominantly dark (melanic) forms during the 19th century. The figure below illustrates the relative visibility of each morph on clean vs. soot-darkened bark.



Which statement CORRECTLY explains industrial melanism as an example of natural selection?

A. The dark melanic form was directly produced (induced) by UV radiation and chemical mutagens released from industrial pollution

B. In polluted (industrial) areas, soot darkened the tree bark; dark moths were better camouflaged and survived bird predation more effectively than pale moths, leading to an increase in dark-morph frequency over generations

C. The dark moth is constitutionally superior to the pale moth in all environments because its greater melanin production provides metabolic advantages

D. Pale moths voluntarily migrated away from industrial areas while dark moths remained, creating a false impression that selection had occurred

Q35. Two key ecological concepts are 'habitat' and 'niche'. Hutchinson (1957) described the niche as an 'n-dimensional hypervolume'. Which statement CORRECTLY distinguishes habitat from ecological niche?

A. Habitat is the functional role of an organism in an ecosystem (what it does); niche is its physical address (where it lives)

B. Two species can permanently coexist in the same ecological niche provided they share the same physical habitat

C. Habitat is the physical location or environment where a species lives; niche encompasses all the biotic and abiotic variables (food resources, temperature range, spatial requirements, interactions) that define

the species' functional role — no two species can share an identical niche indefinitely (Gause's competitive exclusion principle)

D. Niche and habitat are interchangeable ecological terms that represent the same concept

Q36. Cultural eutrophication refers to accelerated nutrient enrichment of water bodies due to human activities such as agricultural run-off and discharge of untreated sewage. Which sequence CORRECTLY describes the chain of events that produces a 'dead zone' (severely hypoxic water body)?

A. Increased dissolved $O_2 \rightarrow$ fish population boom $\rightarrow$ algal growth $\rightarrow$ nutrient enrichment

B. Nutrient enrichment $\rightarrow$ increased dissolved $O_2 \rightarrow$ algal bloom $\rightarrow$ increased fish growth

C. Algal bloom $\rightarrow$ enhanced photosynthesis $\rightarrow$ very high dissolved O_2 $\rightarrow$ rapid aquatic plant growth and fish proliferation

D. Nutrient (N, P) enrichment $\rightarrow$ algal bloom $\rightarrow$ algal death and microbial decomposition $\rightarrow$ O_2 depletion (hypoxia) $\rightarrow$ fish kill

Q37. Biodiversity conservation strategies are classified as in-situ (within the natural habitat) or ex-situ (outside the natural habitat). Which of the following is an example of IN-SITU conservation?

A. Establishment of national parks, wildlife sanctuaries, and biosphere reserves where species are protected within their natural habitats

B. Maintenance of seed banks in temperature- and humidity-controlled facilities for long-term storage of genetic diversity

C. Captive breeding programmes for endangered tigers conducted in zoological parks

D. Cryopreservation of gametes, embryos, or tissue cultures in liquid nitrogen at -196°C

Q38. Ecological succession is the gradual, directional change in community structure over time. Two types are recognised: primary (on bare, previously uncolonised substrate) and secondary (on a previously inhabited area). Which statement CORRECTLY distinguishes secondary succession from primary succession?

A. Secondary succession occurs exclusively in aquatic (limnetic) habitats after primary succession has already occurred on that substrate

B. Secondary succession begins on a substrate that already has a residual soil layer with organic matter and a seed bank from the previous community, making it considerably faster than primary succession

C. Secondary succession can occur only after catastrophic volcanic eruptions that completely destroy all life and the soil profile

D. Secondary succession invariably leads to a grassland climax community regardless of the regional climate or geographical location

Q39. ELISA (Enzyme-Linked Immunosorbent Assay) is a widely used immunological technique for detecting specific antigens or antibodies. In a sandwich ELISA set up to diagnose HIV infection (detecting anti-HIV antibodies in the patient's serum), how is a POSITIVE result indicated?

A. Absence of any colour change in the reaction well, indicating complete antibody saturation and competitive inhibition of the enzyme

B. Visible agglutination (clumping) of red blood cells added to the test sample

C. Development of a measurable colour change (spectrophotometric absorbance signal) because the enzyme conjugated to the secondary antibody cleaves the chromogenic substrate into a coloured product

D. Formation of a visible precipitin line in an agarose gel matrix, indicating antigen–antibody complex precipitation

Q40. RNA interference (RNAi) is a conserved post-transcriptional gene-silencing mechanism in eukaryotes with applications in agriculture (e.g. insect-

resistant crops) and medicine. Which statement CORRECTLY describes the mechanism by which RNAi silences gene expression?

A. RNAi prevents gene transcription by physically blocking RNA polymerase II at the gene's promoter region, preventing transcription initiation

B. RNAi permanently silences genes by methylating their CpG promoter regions and converting chromatin to heterochromatin

C. RNAi works by incorporating siRNA (small interfering RNA) into the ribosome, where it blocks translation initiation by competing with mRNA for the P-site

D. RNAi works by incorporating siRNA into the RISC (RNA-Induced Silencing Complex); the antisense (guide) strand of siRNA directs RISC to complementary target mRNA, which is then cleaved and degraded, silencing the gene

Detailed Solutions

Q1.

Solution

Concept — Gastric Gland Cell Types and HCl Secretion

The gastric mucosa contains four major cell types in its glands: parietal (oxyntic) cells, chief (peptic) cells, G cells, and mucous neck cells. Parietal cells are responsible for secreting HCl into the stomach lumen. HCl is pumped via the H^+/K^+-ATPase (proton pump) located on the apical (luminal) membrane of parietal cells. This active transporter exchanges one H^+ ion out into the lumen for one K^+ ion into the cell, consuming one ATP per cycle and generating the extremely acidic gastric juice (pH $\approx$ 1–2).

Step 1 — Mechanism of H^+/K^+-ATPase:

Inside parietal cells, $CO_2 + H_2O \xrightarrow{CA} H_2CO_3 \rightarrow H^+ + HCO_3^-$. The H^+ is pumped into the lumen via the proton pump, and HCO_3^- exits basally in exchange for Cl^- (the "alkaline tide"). Cl^- is then secreted into the lumen through separate Cl^- channels to maintain electronegativity, combining with H^+ to form HCl.

Why other options are wrong:

Option B: Chief (peptic) cells secrete pepsinogen (the zymogen of pepsin), not HCl; HCl is not a by-product of pepsinogen.

Option C: G cells are enteroendocrine cells that secrete the hormone gastrin into the bloodstream; gastrin indirectly stimulates parietal cells to produce HCl, but G cells themselves do not secrete HCl.

Option D: Mucous neck cells secrete mucus and bicarbonate that protect the stomach wall from acid; they do not produce HCl.

Answer: (A) ← Go back to Q1

Q2.

Solution

Concept — Lactase Deficiency and Lactose Intolerance

Lactase (lactase-phlorizin hydrolase) is a brush-border enzyme expressed on the apical microvilli (brush border) of small intestinal enterocytes, particularly in the jejunum. It hydrolyses lactose (milk sugar) into glucose and galactose for absorption. Deficiency of lactase leads to undigested lactose passing into the colon, where colonic bacteria ferment it, producing gas (CO_2, H_2, CH_4) and short-chain fatty acids, causing the classical symptoms: bloating, flatulence, osmotic diarrhoea, and abdominal cramps.

Step 1 — Location of Lactase:

Lactase is anchored in the apical membrane of small intestinal enterocytes and

is part of the intestinal brush-border enzyme complex. It is NOT secreted into the intestinal lumen; it acts at the surface of the epithelial cells as the digested products come into contact with the brush border.

Step 2 — Primary vs. Secondary Lactase Deficiency:
Primary lactase deficiency (adult-type hypolactasia) is the most common form worldwide, resulting from a genetically programmed decline in lactase expression after weaning. Secondary lactase deficiency can follow intestinal infections or inflammatory bowel disease.

Why other options are wrong:
Option A: Sucrase deficiency causes sucrose intolerance (bloating and diarrhoea after eating sucrose-containing foods like fruit and sweets), not after milk consumption. Moreover, sucrase is also a brush-border enzyme, not a pancreatic enzyme.
Option C: Salivary amylase deficiency would impair initial starch digestion in the mouth but would not cause milk intolerance; salivary amylase has no role in lactose digestion.
Option D: Pepsin deficiency impairs gastric protein digestion, leading to inadequate peptide formation, not lactose intolerance.

Answer: (B) ← Go back to Q2

Q3.

Solution

Concept — CO_2 Transport in Blood: Three Mechanisms
CO_2 produced by tissues is transported to the lungs via three routes: (1) dissolved in plasma (≈7–10%), (2) as carbamino-haemoglobin formed when CO_2 binds the free amino groups (–NH_2) of globin chains (≈20–25%), and (3) as bicarbonate ions (HCO_3^-) in plasma (≈70%). The bicarbonate route is dominant and involves carbonic anhydrase (CA) inside RBCs.

Step 1 — Bicarbonate Formation and Chloride Shift:
Inside RBCs: $CO_2 + H_2O \xrightarrow{CA} H_2CO_3 \rightarrow H^+ + HCO_3^-$. The HCO_3^- rapidly exits the RBC via the Band-3 anion exchanger (AE1) in exchange for Cl^- entering from the plasma. This is the **chloride (Hamburger) shift**. It maintains electrical neutrality inside the RBC and allows further CO_2 loading. H^+ is buffered by deoxyhaemoglobin (which has a higher affinity for H^+ than oxyhaemoglobin).

Step 2 — Reversal in Lungs:
In pulmonary capillaries, the process reverses: Cl^- leaves the RBC, HCO_3^- re-enters, is reconverted to $CO_2 + H_2O$ (by CA), and CO_2 diffuses out of the RBC into alveoli for exhalation.

Why other options are wrong:
Option A: Only ≈7–10% of CO_2 is transported as dissolved CO_2 in plasma (too small to be the 'approximately 70%' route).
Option B: Carbamino-haemoglobin accounts for ≈20–25% of CO_2 transport; CO_2 binds the globin *amino* groups, not the haem group.
Option D: CO_2 does NOT bind to the haem (iron-containing) group of haemoglobin; O_2 binds to the haem iron. CO_2 binds to free amino groups on the protein chains (forming carbamino compounds), and this accounts for only 20–25%, not 50%.

Answer: (C) ← Go back to Q3

Q4.

Solution

Concept — Respiratory Quotient (RQ) for Different Substrates
Respiratory quotient RQ = CO_2 produced / O_2 consumed during aerobic oxidation of a substrate. The value depends on the chemical composition of the fuel: carbohydrates (RQ = 1.0), fats (RQ ≈ 0.7), proteins (RQ ≈ 0.8). Fat molecules are more reduced (higher H:O ratio) than carbohydrates, so proportionally more O_2 is required and less CO_2 is produced per unit energy.

Step 1 — Calculation for Fat (Palmitic Acid Example):
$C_{16}H_{32}O_2 + 23\,O_2 \rightarrow 16\,CO_2 + 16\,H_2O$
RQ = 16/23 ≈ **0.70**. For a typical mixed fat, RQ ≈ 0.7.

Step 2 — Physiological Interpretation:
During prolonged fasting or a strictly carbohydrate-free diet, the body shifts to β-oxidation of fatty acids. The RQ therefore falls toward 0.7, which can be measured in a metabolic chamber. An RQ < 0.7 would indicate lipogenesis from protein, which is unusual.

Why other options are wrong:
Option A: RQ = 1.0 is characteristic of carbohydrate (glucose) oxidation: $C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O$; not applicable when only fat is being oxidised.
Option B: RQ ≈ 0.8 corresponds to protein catabolism or a mixed diet; not applicable here.
Option C: RQ > 1.0 (e.g., 1.2–1.4) occurs only during active conversion of carbohydrates to fat (lipogenesis), releasing more CO_2 than O_2 consumed. This is not the scenario described.

Answer: (D) ← Go back to Q4

Q5.

Solution

Concept — Rh Incompatibility and Erythroblastosis Fetalis

Erythroblastosis fetalis (haemolytic disease of the newborn, HDN) occurs when an Rh-negative mother is sensitised to the Rh (D) antigen present on fetal RBCs during delivery or abortion of an Rh-positive fetus. She produces anti-D IgG antibodies (sensitisation). In a subsequent Rh-positive pregnancy, maternal anti-D IgG crosses the placenta and destroys fetal RBCs by haemolysis. The fetus responds by releasing immature erythroblasts (nucleated RBCs) into circulation — hence the name.

Step 1 — Mechanism:

Anti-D IgG (but not IgM) crosses the placental syncytiotrophoblast via Fc receptors. In the fetal circulation, anti-D coats fetal Rh^+ RBCs $\rightarrow$ destruction by fetal macrophages and complement $\rightarrow$ severe haemolytic anaemia, jaundice (kernicterus from bilirubin), hydrops fetalis.

Step 2 — Prevention:

Anti-D immunoglobulin (RhoGAM) is given to Rh-negative mothers at 28 weeks gestation and within 72 hours of delivery of an Rh-positive baby. RhoGAM destroys any fetal RBCs that enter the maternal circulation before they can sensitise the mother's immune system.

Why other options are wrong:

Option B: Thalassaemia is a hereditary haemoglobinopathy caused by mutations in globin-chain genes; it is not caused by alloimmunisation or maternal antibodies.

Option C: Polycythaemia vera is a myeloproliferative neoplasm with excessive RBC production; it is unrelated to Rh incompatibility.

Option D: Sickle-cell crisis is caused by polymerisation of HbS (a point mutation Glu$\rightarrow$Val in β-globin) under hypoxic conditions; it is not caused by maternal antibodies.

Answer: (A) ← Go back to Q5

Q6.

Solution

Concept — Cardiac Pacemaker: The Sinoatrial (SA) Node

The SA node is a small collection of specialised pacemaker cells located in the superior wall of the right atrium near the opening of the superior vena cava. It is called the pacemaker because it spontaneously depolarises at the *highest intrinsic rate* ($\approx$70–75 bpm in adults) compared to other potential pacemakers (AV node $\approx$40–55 bpm; Purkinje fibres $\approx$20–40 bpm). Because the SA node fires first, it suppresses the intrinsic automaticity of all downstream pacemakers (overdrive

suppression) and imposes its rate on the entire heart.

Step 1 — Conduction Pathway:
SA node → atrial myocardium (P-wave on ECG) → AV node (delay of ≈120 ms, PR interval) → Bundle of His → right and left bundle branches → Purkinje fibres → ventricular myocardium (QRS complex).

Step 2 — Neural Modulation:
The SA node rate is modulated by the autonomic nervous system (sympathetic ↑ rate; parasympathetic/vagus ↓ rate) but retains intrinsic automaticity even when denervated.

Why other options are wrong:
Option A: The SA node is NOT the largest structure; it is a small (≈1.5 cm) cluster of cells. Size is not the criterion for pacemaker function.
Option C: The SA node does receive autonomic input (via cardiac accelerator and vagus nerves), but it initiates impulses intrinsically (due to the funny current, If, and low resting K^+ conductance), not by direct command from the medulla.
Option D: The SA node is located in the right atrial wall (not the interventricular septum). The AV node is near the interatrial septum; the Bundle of His runs through the membranous ventricular septum.

Answer: (B) ← Go back to Q6

Q7.

Solution

Concept — Hepatic Portal System: Nutrient Transport to the Liver
The hepatic portal system is a specialised venous circuit that drains blood from the stomach, small intestine, large intestine, pancreas, and spleen and delivers it to the liver before it enters the systemic (general) circulation. The hepatic portal vein is formed by the union of the superior mesenteric vein (draining the small intestine and proximal colon) and the splenic vein. It carries absorbed nutrients (glucose, amino acids, water-soluble vitamins, short-chain fatty acids) directly to hepatic sinusoids for first-pass metabolism, storage, or redistribution.

Step 1 — Why 'First Pass' Matters:
The liver detoxifies drugs and metabolises many substances before they reach systemic circulation. Oral drugs with high first-pass extraction (e.g., morphine, propranolol) must be given in larger oral doses than intravenous doses because much is metabolised in the liver before reaching the bloodstream.

Why other options are wrong:
Option A: The hepatic artery branches from the coeliac trunk (a branch of the aorta) and delivers oxygenated blood TO the liver for its metabolic needs; it does

not carry absorbed intestinal nutrients.
Option B: The hepatic veins (right, middle, left) carry blood AWAY from the liver into the inferior vena cava; they transport blood that has already been processed by the liver.
Option D: The superior mesenteric artery is an ARTERIAL vessel that supplies oxygenated blood to the small intestine and parts of the large intestine; blood flows into, not out of, the intestine via this route.

Answer: (C) ← Go back to Q7

Q8.

Solution

Concept — Ornithine (Urea) Cycle: First Committed Step and OTC
The ornithine cycle (Krebs–Henseleit cycle) detoxifies ammonia (NH_3) in the liver by incorporating it into urea, which is excreted in urine. The cycle has five steps. The preparatory step (outside the cycle) is the synthesis of carbamoyl phosphate from NH_3 + CO_2 + 2ATP by **carbamoyl phosphate synthetase I (CPS-I)** in the mitochondrial matrix.

Step 1 — First Committed Step Inside the Cycle (OTC):
The first committed step *within* the cycle is the condensation of ornithine with carbamoyl phosphate to form citrulline, catalysed by **ornithine transcarbamylase (OTC)**, also located in the mitochondrial matrix. This step is considered the 'committed step' of the cycle because citrulline is an intermediate specific to the urea cycle and is exported to the cytoplasm for subsequent steps.

Step 2 — Remaining Steps:
Cytoplasmic steps: Citrulline + Aspartate $\xrightarrow{\text{ASS}}$ Argininosuccinate $\xrightarrow{\text{ASL}}$ Arginine + Fumarate $\xrightarrow{\text{Arginase}}$ Urea + Ornithine. Ornithine re-enters the mitochondrion to complete the cycle.

Why other options are wrong:
Option A: Arginase catalyses the *last* step of the cycle — the hydrolysis of arginine to produce urea + ornithine — not the first committed step.
Option B: Argininosuccinate lyase (ASL) catalyses the *third* step inside the cycle — the cleavage of argininosuccinate to form arginine and fumarate.
Option C: Carbamoyl phosphate synthetase I (CPS-I) catalyses the preparatory step (synthesis of carbamoyl phosphate) that feeds INTO the cycle, but the condensation of ornithine with this carbamoyl phosphate (forming citrulline) is the first committed step of the cycle itself, and this is catalysed by OTC, not CPS-I.

Answer: (D) ← Go back to Q8

Q9.

Solution

Concept — Uricotelic Organisms: Nitrogenous Waste Excretion

Animals excrete nitrogenous waste in three main forms: (1) **Ammonotelic** — excrete ammonia (NH_3): most aquatic invertebrates, bony fishes (teleosts like salmon), and aquatic larval amphibians. Ammonia is highly toxic but soluble and easily diluted in water. (2) **Ureotelic** — excrete urea: mammals, cartilaginous fishes (sharks), and *adult* amphibians. Urea is less toxic than NH_3 but requires water for excretion. (3) **Uricotelic** — excrete uric acid: reptiles (lizards, snakes, tortoises), birds, and insects. Uric acid is nearly insoluble, semi-solid, and non-toxic; it requires minimal water — an adaptation for terrestrial life and/or shelled eggs.

Step 1 — Why Birds are Uricotelic:

Birds (like the pigeon, *Columba livia*) excrete uric acid as white, pasty nitrogenous waste (seen in bird droppings). Uric acid production conserves water — critical for flight and for developing embryos inside shelled eggs (where urea would become toxic).

Why other options are wrong:

Option B: Adult frogs are **ureotelic** (they switch from ammonotelic tadpoles to ureotelic adults upon metamorphosis; living on land requires a less toxic, more concentrated waste product than ammonia).

Option C: Salmon (*Salmo salar*) is a bony fish (teleost) and is **ammonotelic** — it excretes ammonia directly across its gills into the surrounding water.

Option D: Earthworms (*Pheretima*) are **ammonotelic** — they excrete ammonia through their moist body surface and nephridia, relying on their moist soil environment to dilute the toxic waste.

Answer: (A) ← Go back to Q9

Q10.

Solution

Concept — Glomerular Filtration Barrier: Exclusion of Plasma Proteins

The glomerular filtration barrier has three layers: (1) the fenestrated capillary endothelium (pores ≈70–100 nm), (2) the glomerular basement membrane (GBM) composed of type IV collagen, laminin, and negatively charged heparan sulphate proteoglycans, and (3) the podocyte slit diaphragms (filtration slits ≈4–11 nm wide). The barrier restricts passage of macromolecules by two mechanisms: size selectivity (molecules >8 nm radius are excluded) and charge selectivity (polyanions like albumin are repelled by the negatively charged GBM).

Step 1 — Why Albumin is Excluded:

Serum albumin ($M_r \approx 69{,}000$ Da; radius ≈3.5 nm; net charge strongly negative at

physiological pH) is excluded primarily by the *negative charge* of the GBM proteoglycans (electrostatic repulsion) and partially by the slit-diaphragm size barrier. In conditions that destroy the negative charge (e.g., nephrotic syndrome), massive proteinuria occurs even though the molecular radius alone might not exclude albumin.

Why other options are wrong:
Option A: Tubular secretion moves substances FROM blood INTO the tubular lumen (e.g., PAH, penicillin); the reverse (from lumen back to blood) is called *reabsorption*. Small amounts of filtered proteins are reabsorbed by receptor-mediated endocytosis in the PCT, but this does not explain *why* they are absent from the filtrate in the first place.
Option C: Albumin is actually *abundant* in plasma ($\approx$4 g/dL, the most plentiful plasma protein); its absence from the filtrate is due to the filtration barrier, not low concentration.
Option D: Plasma proteins do not bind to glucose. Glucose is freely filtered and actively reabsorbed in the PCT by SGLT transporters; the absence of proteins from the filtrate is unrelated to glucose transport.

Answer: (B) ← Go back to Q10

Solution

Concept — Sarcomere Structure and Changes During Contraction (Sliding Filament Theory)
A sarcomere (Z-line to Z-line) contains: thick myosin filaments (entire A-band) and thin actin filaments (I-band through part of A-band). Key bands and their behaviour during contraction:

Q11.

- **I-band** (light): contains only thin (actin) filaments. **DECREASES** during contraction as thin filaments slide further into the A-band.
- **H-zone** (lighter centre of A-band): contains only thick (myosin) filaments where actin does not overlap. **DECREASES** as actin filaments slide in and cover more of the H-zone.
- **A-band** (dark): spans the entire length of thick myosin filaments. **REMAINS CONSTANT** (myosin filaments do not change length during contraction).
- **M-line** and **Z-lines**: structural anchors; Z-lines move closer together as the sarcomere shortens.

Step 1 — Sliding Filament Mechanism:
Myosin cross-bridges attach to actin, undergo a power stroke pulling actin filaments toward the M-line. Neither myosin nor actin filaments change in length;

only the degree of overlap changes. The sarcomere shortens ⇒ I-bands narrow, H-zone narrows, A-band constant.

Why other options are wrong:
Option A: The A-band does NOT decrease; it remains constant because it equals the length of the myosin filament, which does not shorten.
Option B: The A-band stays constant; saying both A-band and I-band decrease is incorrect.
Option D: The H-zone alone is incomplete; both the I-band AND the H-zone decrease simultaneously during contraction.

Answer: (C) ← Go back to Q11

Q12.

Solution

Concept — Role of Ca^{2+} in Skeletal Muscle Contraction: Troponin–Tropomyosin Regulation
At rest, the regulatory protein complex (troponin I, T, C + tropomyosin) keeps tropomyosin physically blocking the myosin-binding sites on actin. When a motor neuron fires, acetylcholine is released at the neuromuscular junction, triggering an action potential in the sarcolemma that propagates into the T-tubules, causing Ca^{2+} release from the sarcoplasmic reticulum (SR).

Step 1 — Ca^{2+} Action:
Ca^{2+} ($\approx 10^{-5}$ M in activated cytoplasm) binds to **troponin C** (the calcium-binding subunit of the troponin complex). This induces a conformational change in troponin I and T, which causes **tropomyosin to shift** laterally by ≈7–8 Å on the actin filament. This movement exposes the myosin-binding sites on actin, allowing cross-bridge formation and the power stroke.

Step 2 — Cross-Bridge Cycle:
Myosin head (charged with ADP + P_i) binds actin → power stroke (ADP + P_i released, myosin pulls actin toward M-line) → ATP binds → myosin detaches from actin → ATP is hydrolysed to ADP + P_i → myosin re-cocks. Cycle repeats as long as Ca^{2+} is elevated.

Why other options are wrong:
Option A: Ca^{2+} is not an energy source or electron donor. Energy for contraction comes from ATP hydrolysis by myosin ATPase (activated by the actin–myosin interaction).
Option B: ATP is hydrolysed by the myosin ATPase activity of the myosin head itself (upon binding actin), not by Ca^{2+}.
Option C: Ca^{2+} is released *inside* the muscle fibre from the SR; it does not act at the neuromuscular junction to release acetylcholine (that is the role of Ca^{2+} at the

motor nerve terminal, which is a separate pool of extracellular Ca^{2+}).

Answer: (D) ← Go back to Q12

Q13.

Solution

Concept — Functions of the Cerebellum

The cerebellum is responsible for: (1) **coordination of voluntary movements** (smooth, precise execution), (2) **maintenance of balance and posture** via integration of proprioceptive, vestibular, and visual inputs, (3) **motor learning** (adaptation and refinement of movements over time), and (4) timing of rapid alternating movements. The cerebellum does NOT initiate voluntary movement (that is the cerebral motor cortex) and is NOT involved in conscious sensation or memory formation.

Step 1 — Clinical Features of Cerebellar Damage:

Classic cerebellar syndrome includes: **ataxia** (unsteady, staggering gait), **dysmetria** (inability to gauge distance — past-pointing), **dysdiadochokinesia** (inability to rapidly alternate movements, e.g. pronation/supination), **intention tremor** (tremor that worsens as the hand approaches a target), and nystagmus (abnormal eye movements). Speech becomes dysarthric (slurred, scanning speech).

Why other options are wrong:

Option B: Loss of conscious somatic sensation (touch, pain, proprioception) from the contralateral body would result from damage to the thalamus or somatosensory cortex (post-central gyrus of the parietal lobe), not the cerebellum.

Option C: Inability to form new explicit memories (anterograde amnesia) is caused by hippocampal damage (e.g. bilateral hippocampectomy, as in patient H.M., or Korsakoff syndrome). The cerebellum is involved in procedural motor learning, not declarative memory.

Option D: Voluntary respiratory rhythm is generated by the pre-Bötzinger complex and respiratory groups in the medulla oblongata and pons; cerebellar damage does not abolish voluntary breathing.

Answer: (A) ← Go back to Q13

Q14.

Solution

Concept — Saltatory Conduction in Myelinated Nerve Fibres

In unmyelinated axons, the action potential propagates continuously along the entire membrane surface as each adjacent patch of membrane depolarises in turn (continuous conduction). In myelinated axons, the myelin sheath (formed by Schwann cells in the PNS, oligodendrocytes in the CNS) electrically insulates the

internodal membrane, preventing ion flow across it. Voltage-gated Na^+ and K^+ channels are concentrated only at the **nodes of Ranvier** (the unmyelinated gaps between myelin segments, ≈1–2 μm wide).

Step 1 — Mechanism of Saltatory Conduction:
Depolarisation at one node generates a local current that 'jumps' to the next node (skipping the myelinated internodal segment), where voltage-gated channels regenerate the action potential. This node-to-node jumping is saltatory conduction (Latin: *saltare* = to leap). It is faster because: (a) the electrical signal jumps across long internodal distances (≈1 mm) rather than propagating 1 μm at a time; (b) fewer regions of membrane need to depolarise/repolarise, reducing metabolic cost.

Step 2 — Conduction Velocity:
Large myelinated axons (e.g. Aα fibres, ≈20 μm diameter) conduct at 70–120 m/s. Small unmyelinated C fibres (≈0.2–1.5 μm) conduct at only 0.5–2 m/s.

Why other options are wrong:
Option A: Myelin sheaths do NOT contain voltage-gated Na^+ channels — that is the whole point. Channels are at the nodes, and the myelin acts as an insulator between nodes.
Option C: Myelinated fibres actually have a *larger* axonal diameter than unmyelinated fibres of comparable conduction speed. Larger diameter *reduces* internal (axial) resistance, further increasing conduction velocity, but the primary advantage of myelin is saltatory conduction.
Option D: The myelin sheath is an electrical insulator (lipid-rich membrane); it does not secrete neurotransmitters. Neurotransmitters are released at synaptic terminals.

Answer: (B) ← Go back to Q14

Q15.

Solution

Concept — Insulin Signalling and GLUT-4 Translocation
Insulin lowers blood glucose primarily by stimulating glucose uptake into skeletal muscle and adipose tissue. These tissues express GLUT-4 (glucose transporter type 4), which is unique in being **insulin-responsive**: at rest, GLUT-4 is stored in intracellular vesicles (GSVs, GLUT4-storage vesicles); upon insulin signalling, these vesicles fuse with the plasma membrane, dramatically increasing surface GLUT-4 density and glucose import capacity.

Step 1 — Insulin Signalling Cascade:
Insulin binds receptor (receptor tyrosine kinase) → autophosphorylation of receptor → IRS-1/2 phosphorylation → PI3K activation → PIP3 production → PDK1

activation → Akt (PKB) phosphorylation → AS160 phosphorylation → Rab GTPase activation → **GLUT-4 vesicle translocation to plasma membrane**.

Step 2 — Tissues and Transporter Types:
GLUT-1: ubiquitous (basal glucose uptake). GLUT-2: liver, pancreatic β-cells (glucose sensor). GLUT-3: neurons. **GLUT-4**: skeletal muscle, cardiac muscle, adipose (insulin-regulated). GLUT-5: fructose transport in intestine.

Why other options are wrong:
Option A: Insulin does not form its own glucose channel. It signals GLUT-4 transporters (pre-existing proteins) to move to the surface.
Option B: Insulin *inhibits* glycogen phosphorylase (which breaks down glycogen) and *activates* glycogen synthase (which builds glycogen). Insulin promotes glycogen synthesis and glucose storage, not glycogenolysis.
Option D: Glucokinase (hexokinase IV) is activated by insulin in *liver* hepatocytes, not in skeletal muscle or adipose cells. In these peripheral tissues, glucose uptake depends on GLUT-4 insertion into the plasma membrane, not glucokinase activity.

Answer: (C) ← Go back to Q15

Q16.

Solution

Concept — Parathyroid Hormone (PTH) and Calcium Homeostasis
PTH is secreted by the four parathyroid glands (embedded in the posterior thyroid) in response to falling plasma Ca^{2+}. PTH raises blood Ca^{2+} via three mechanisms: (1) **bone**: stimulates osteoclast activity (bone resorption, releasing Ca^{2+} and PO_4^{3-}); (2) **kidney**: increases Ca^{2+} reabsorption in the distal convoluted tubule (DCT) and decreases phosphate reabsorption (phosphaturia); (3) **intestine**: stimulates renal conversion of 25(OH)D to 1,25$(OH)_2$D (calcitriol), which increases intestinal Ca^{2+} absorption.

Step 1 — Hypoparathyroidism (Low PTH):
Without PTH: (a) no stimulation of bone resorption $\Rightarrow$ Ca^{2+} not released from bone; (b) no renal Ca^{2+} retention $\Rightarrow$ urinary Ca^{2+} decreases; (c) no calcitriol production $\Rightarrow$ intestinal Ca^{2+} absorption falls. Net result: **low blood Ca^{2+}** (hypocalcaemia). Low Ca^{2+} increases neuronal membrane excitability (by reducing the threshold for action potential firing) $\Rightarrow$ **muscle tetany, perioral tingling, carpopedal spasm (Trousseau's sign), seizures**.

Why other options are wrong:
Option A: High blood Ca^{2+} and increased osteoclast activity describe *hyperparathyroidism* (too much PTH), the opposite of hypoparathyroidism. Low phosphate also describes hyperparathyroidism (PTH promotes phosphate excretion).
Option B: Increased bone resorption again describes *excess* PTH, not its absence.

In hypoparathyroidism, bone resorption is reduced.
Option C: Normal Ca^{2+} with compensatory urinary Ca^{2+} loss does not describe hypoparathyroidism. In fact, urinary Ca^{2+} loss would *decrease* in hypoparathyroidism because less Ca^{2+} is filtered (less circulates) and renal reabsorption is unchanged.

Answer: (D) ← Go back to Q16

Q17.

Solution

Concept — Ribosome Structure: Prokaryotic (70S) vs. Eukaryotic (80S)
Ribosomes are ribonucleoprotein complexes that catalyse mRNA-directed peptide bond formation (protein synthesis). Their sedimentation coefficients (Svedberg units, S) differ between prokaryotes and eukaryotes. Note: S values are not additive (they reflect both size and shape).

Step 1 — Prokaryotic Ribosomes (70S):
Total 70S = **30S small subunit** (16S rRNA + ≈21 proteins) + **50S large subunit** (23S + 5S rRNA + ≈31 proteins). The 30S subunit decodes mRNA; the 50S catalyses peptide bond formation (peptidyl transferase activity is in the 23S rRNA — a ribozyme).

Step 2 — Eukaryotic Cytoplasmic Ribosomes (80S):
Total 80S = **40S small subunit** (18S rRNA + ≈33 proteins) + **60S large subunit** (28S + 5.8S + 5S rRNA + ≈49 proteins). Note: mitochondrial and chloroplast ribosomes are 70S (reflecting endosymbiotic origin).

Step 3 — Clinical Significance:
Antibiotics targeting 30S (tetracyclines, aminoglycosides) or 50S (chloramphenicol, erythromycin, linezolid) selectively inhibit prokaryotic protein synthesis without affecting eukaryotic 80S ribosomes.

Why other options are wrong:
Option B: This has the sizes reversed — prokaryotes are 70S, not 80S; eukaryotes are 80S, not 70S.
Option C: The 70S prokaryotic ribosome has a **30S** small subunit and a **50S** large subunit; 60S + 40S are the eukaryotic subunits. These are misassigned in option C.
Option D: Prokaryotic ribosomes are 70S, not 80S. Both having 80S would make them indistinguishable and antibiotic selectivity would be impossible.

Answer: (A) ← Go back to Q17

Q18.

Solution

Concept — Smooth ER: Steroid Synthesis and Drug Detoxification

The smooth endoplasmic reticulum (sER) is an extensive membrane network lacking ribosomes. Its key functions include: (1) **lipid and phospholipid synthesis**, (2) **steroid hormone biosynthesis** (using cholesterol via CYP enzymes), (3) **drug/toxin detoxification** via cytochrome P450 (CYP) monooxygenase enzymes (particularly CYP3A4 in hepatocytes), (4) calcium ion storage and release (in muscle, as the sarcoplasmic reticulum), and (5) carbohydrate metabolism (glucose-6-phosphatase for glycogenolysis).

Step 1 — Why sER is Abundant in Steroid-Producing Cells:

Steroid hormones (oestrogen, testosterone, cortisol, aldosterone) are synthesised from cholesterol through a series of CYP enzyme reactions on the sER membrane. Cells that produce large quantities of steroids (adrenal cortex, Leydig cells of testis, liver hepatocytes for bile acids) accordingly have extensive sER networks.

Step 2 — Drug Detoxification by CYP450:

Hepatocyte CYP450 enzymes hydroxylate lipophilic drugs and toxins, making them more polar and water-soluble for urinary excretion (Phase I metabolism). These enzymes are embedded in the sER membrane.

Why other options are wrong:

Option A: Steroid synthesis is a *lipid*-based process that does NOT require ribosomes. It is an enzymatic process on the sER membrane. The rough ER (with ribosomes) makes secretory and membrane proteins, not steroids.

Option C: Drug detoxification (CYP450 hydroxylation) occurs in the smooth ER, not the rough ER. Rough ER is for protein secretion.

Option D: While both types of ER can coexist in hepatocytes, the specific activities described (steroid synthesis and CYP450 drug metabolism) are exclusively sER functions, making sER the MOST abundant organelle in a cell maximally engaged in these processes.

Answer: (B) ← Go back to Q18

Q19.

Solution

Concept — Cell Cycle Checkpoints: G2/M Checkpoint

The cell cycle has three major checkpoints: (1) **G1/S (restriction) checkpoint**: assesses cell size, nutrient availability, growth factor signalling, and DNA integrity; if DNA is damaged, p53 activates p21, which inhibits CDK2/Cyclin E, halting the cell in G1. (2) **G2/M checkpoint**: assesses whether DNA replication (S phase) is complete and accurate before entry into mitosis. Incompletely replicated or damaged DNA activates ATM/ATR kinases, which phosphorylate Chk1/2, inhibiting

Cdc25C phosphatase, thus keeping CDK1/Cyclin B inactive (preventing mitotic entry). (3) **Spindle assembly checkpoint (SAC)**: during M phase, monitors for proper kinetochore–microtubule attachment at all chromosomes before anaphase onset.

Step 1 — G2/M Checkpoint Specifically:
The G2/M checkpoint ensures that: (a) every base pair of DNA has been replicated exactly once; (b) no double-strand breaks or mismatches remain; (c) sufficient cyclin B has accumulated to drive mitotic entry. Only after these conditions are met is CDK1/Cyclin B (the Maturation Promoting Factor, MPF) fully activated, triggering nuclear envelope breakdown and chromosome condensation.

Why other options are wrong:
Option A: The G1/S checkpoint (restriction point) monitors for DNA damage and cell readiness *before* DNA replication begins. It does not assess whether replication has been completed correctly (that is the G2/M checkpoint's role).
Option B: The spindle assembly checkpoint (SAC) operates *inside* mitosis (metaphase) and monitors kinetochore–microtubule tension. It prevents premature anaphase, not premature mitotic entry after incomplete S phase.
Option D: The SAC also serves as the metaphase-to-anaphase transition checkpoint. There is no separate, formally named "G1 DNA-damage checkpoint" distinct from the G1/S restriction point; the wording overlaps and the G2/M checkpoint is the correct answer for monitoring *completion* of DNA replication.

Answer: (C) ← Go back to Q19

Q20.

Solution

Concept — Chromosomal Theory of Inheritance: Sutton and Boveri (1902–1904)
Walter Sutton (1902) studying grasshopper (*Brachystola magna*) chromosomes during meiosis, and Theodor Boveri (1902–1904) studying sea urchin embryos with abnormal chromosome numbers, independently proposed that chromosomes are the physical carriers of Mendel's hereditary factors (genes). Their key observations: chromosomes come in pairs (like Mendel's factor pairs); homologous chromosomes separate during meiosis I (like allele segregation); non-homologous chromosomes assort independently during meiosis I (like Mendel's independent assortment in the dihybrid cross).

Step 1 — Significance:
The chromosomal theory provided the first cytological, material basis for Mendel's laws, demonstrating that hereditary particles exist on physical structures (chromosomes) that behave in ways that exactly parallel Mendel's mathematical ra-

tios. Morgan's subsequent work with *Drosophila* (1910–1915) experimentally confirmed the theory by demonstrating sex-linked inheritance and gene linkage.

Why other options are wrong:
Option A: X-ray diffraction of DNA was performed by Rosalind Franklin and Maurice Wilkins in the 1950s; Watson and Crick used this data to solve the double helix structure in 1953. Sutton and Boveri worked with light microscopy on chromosomes, not X-ray diffraction.
Option B: We now know definitively (via Avery, Hershey–Chase, and the double helix) that genes are made of DNA, not protein. Sutton–Boveri showed chromosomes carry genes but did not determine the chemical nature of genes.
Option C: Mutation as a DNA sequence change was established much later with molecular biology; Sutton and Boveri worked at the chromosomal, not molecular, level.

Answer: (D) ← Go back to Q**20**

Q21.

Solution

Concept — Test Cross: Identifying Genotype from Offspring Ratio
A test cross (back cross) is the cross of an individual with a *dominant phenotype* (unknown genotype, either FF or Ff) with a *homozygous recessive* individual (ff). The offspring phenotype ratio reveals the genotype of the dominant parent.

Step 1 — Interpreting the 50:50 Ratio:
If parent is Ff: $Ff \times ff \rightarrow \frac{1}{2}\,Ff$ (purple) $+\frac{1}{2}\,ff$ (white) = **50% purple : 50% white**. This matches the observation.
If parent is FF: $FF \times ff \rightarrow$ all Ff (purple) = **100% purple : 0% white**. This does NOT match.
Therefore, the purple-flowered parent must be Ff (heterozygous).

Step 2 — Diagnostic Power of the Test Cross:
The test cross is diagnostic precisely because the homozygous recessive parent contributes only f alleles, so the offspring phenotype directly reveals which allele the dominant parent contributed. A 1:1 ratio unambiguously identifies heterozygosity.

Why other options are wrong:
Option B: If the parent were FF, ALL offspring from a test cross ($FF \times ff$) would be Ff (purple). A 50:50 ratio is impossible if the parent is homozygous dominant.
Option C: ff is homozygous recessive. An ff individual would show the *white* (recessive) phenotype, not purple. The parent in this question displays purple (dominant phenotype), so ff is impossible.
Option D: A 50:50 ratio from a test cross *unambiguously* identifies the parent as

heterozygous (Ff). Additional experiments are not needed; this is the standard genetic test. One cannot argue statistical fluctuation can explain 50:50 — that is the expected Mendelian ratio for a heterozygote, not a fluke.

Answer: (A) ← Go back to Q21

Q22.

Solution

Concept — X-linked Recessive Inheritance: Haemophilia A

Haemophilia A (factor VIII deficiency) is X-linked recessive. The gene encoding factor VIII is on the X chromosome. Males are hemizygous (X^HY = unaffected; X^hY = haemophilic). Females need two copies of the recessive allele to be affected (X^hX^h = haemophilic); carrier females (X^HX^h) are phenotypically normal but transmit the allele to offspring.

Step 1 — Cross: Carrier Mother × Unaffected Father:

Mother: X^HX^h (carrier); Father: X^HY (unaffected).

Possible sons: X^HY (unaffected) or X^hY (haemophilic) — each with probability 1/2.

Possible daughters: X^HX^H (unaffected, non-carrier) or X^HX^h (carrier) — neither haemophilic.

Therefore, the probability that any **son** is haemophilic = **50%** (1 in 2 sons).

Step 2 — Why Daughters are Unaffected:

The father contributes X^H to all daughters. So daughters are either X^HX^H (normal) or X^HX^h (carrier). No daughters will be haemophilic from this cross.

Why other options are wrong:

Option A: 25% would be the probability for *any child* (son or daughter) to be haemophilic ($\frac{1}{4}$ of all offspring from this cross are X^hY), not specifically for *sons*. Among sons only, the probability is 50%.

Option C: 75% is impossible; it would require the majority of sons to receive the haemophilia allele, which is not the case in this cross.

Option D: Sons of carrier mothers absolutely CAN be haemophilic. Sons receive their X chromosome from their *mother*, not their father. The unaffected father contributes only Y to his sons; the mother can contribute either X^H or X^h to her sons.

Answer: (B) ← Go back to Q22

Q23.

Solution

Concept — Down Syndrome: Trisomy 21 and Non-disjunction

Down syndrome (trisomy 21; 2n+1 = 47 chromosomes) is the most common chromosomal disorder, affecting ≈1 in 700–800 live births. In ≈95% of cases, the extra chromosome 21 arises from **non-disjunction** — the failure of chromosome 21 homologs (or sister chromatids) to separate during meiosis I or meiosis II in one parent, most often the mother. This results in a disomic gamete (with 2 copies of chr 21) which, upon fertilisation with a normal gamete (1 copy of chr 21), produces a trisomic zygote (3 copies of chr 21 = trisomy 21).

Step 1 — Clinical Features:

Intellectual disability (mild to moderate), hypotonia, characteristic facial features (oblique palpebral fissures, epicanthal folds, small nose, protruding tongue), single palmar crease (simian line), Brushfield spots (iris speckling), congenital heart defects (≈40–50%), increased risk of leukaemia, and Alzheimer's disease by middle age. Non-disjunction risk increases with maternal age.

Step 2 — Other Forms (Minority):

≈4–5% of Down syndrome cases are due to Robertsonian translocation (chr 21 attached to chr 14 or chr 13); ≈1% are mosaic (non-disjunction in early mitosis, not meiosis).

Why other options are wrong:

Option A: Monosomy 21 (45,XX/XY,-21) is generally not viable; it leads to very early embryonic loss and is not associated with Down syndrome.

Option B: Trisomy 18 is **Edwards syndrome**, not Down syndrome. It presents with severe intellectual disability, clenched fists with overlapping fingers, rocker-bottom feet, and congenital heart defects; it is more severe and usually fatal within the first year.

Option D: A deletion of the long arm of chromosome 21 (del(21q)) would result in partial monosomy 21, which is a different and rarer syndrome; it does not produce the typical Down syndrome phenotype.

Answer: (C) ← Go back to Q23

Q24.

Solution

Concept — tRNA Structure: Anticodon Loop and Codon Recognition

Transfer RNA (tRNA) functions as the adaptor molecule in translation, linking the mRNA codon to the correct amino acid. Each tRNA has a cloverleaf secondary structure with four major regions: (1) **Acceptor stem** (3'-CCA-OH): amino acid covalently attached here by aminoacyl-tRNA synthetase. (2) **D-loop** (dihydrouridine loop): recognised by aminoacyl-tRNA synthetase for identity element match-

ing. (3) **TψC loop** (TpsiC loop): interacts with the ribosome (EF-Tu in prokaryotes, eEF1A in eukaryotes) and helps accommodate tRNA in the ribosomal A-site. (4) **Anticodon loop**: contains the three-nucleotide **anticodon**, which is complementary and antiparallel to the mRNA codon; this is the codon-reading region.

Step 1 — Anticodon–Codon Base Pairing:
During translation at the ribosome, the tRNA anticodon (3'→5') base-pairs with the mRNA codon (5'→3') in the A-site of the small ribosomal subunit. This Watson–Crick (and Wobble) base pairing ensures the correct amino acid is incorporated. The anticodon loop in humans occupies positions 34–36 of the 76-nucleotide tRNA.

Why other options are wrong:
Option A: The acceptor stem (3'-CCA-OH) is the site of amino acid attachment (aminoacylation). It does not base-pair with the mRNA; it interacts with aminoacyl-tRNA synthetase and, during peptide bond formation, with the peptidyl transferase centre of the large ribosomal subunit.
Option B: The D-loop (dihydrouridine loop) contains modified nucleotides (dihydrouridine) and is a major identity element recognised by specific aminoacyl-tRNA synthetases; it does not base-pair with mRNA.
Option C: The TψC loop (containing pseudouridine, ψ) mediates tRNA binding to the ribosome via interaction with elongation factors and the ribosomal surface; it does not base-pair with mRNA codons.

Answer: (D) ← Go back to Q24

Q25.

Solution

Concept — The *trp* Operon: Repressible Operon Regulation
The *trp* operon of *E. coli* controls five structural genes (*trpE, trpD, trpC, trpB, trpA*) encoding enzymes for tryptophan biosynthesis. It is a **repressible operon** (negative control): transcription is normally ON (when tryptophan is low) and is turned OFF when tryptophan is abundant.

Step 1 — Molecular Mechanism (High Trp):
When intracellular tryptophan is high: tryptophan (the **corepressor**) binds to the trp repressor protein (aporepressor), changing its conformation to the **active repressor** form. The active repressor–tryptophan complex binds to the **operator** DNA sequence (overlapping with the promoter), physically blocking RNA polymerase from transcribing the trp structural genes. Result: tryptophan biosynthesis is turned OFF (feedback inhibition at the transcriptional level).

Step 2 — Contrast with lac Operon:
The *lac* operon is **inducible** (OFF by default; the inducer allolactose inactivates the

lac repressor, turning genes ON). The *trp* operon is **repressible** (ON by default; the corepressor tryptophan activates the repressor, turning genes OFF). Additionally, the trp operon has attenuation (leader sequence acts as an RNA thermometer for ribosome stalling depending on Trp availability) as a secondary regulatory mechanism.

Why other options are wrong:
Option B: Tryptophan does NOT bind directly to the operator DNA. The binding is mediated by the trp repressor protein; tryptophan changes the repressor's conformation, and it is the repressor that binds the operator.
Option C: The trp operon is *repressible*, not inducible. High tryptophan turns transcription *OFF*, not ON. If tryptophan induced the operon, the cell would make more and more tryptophan without limit — the opposite of metabolic efficiency.
Option D: Tryptophan *activates* (not inactivates) the trp repressor. An inactive repressor cannot bind the operator; the operon would then remain ON. The corepressor model requires tryptophan to activate the repressor so that it can bind the operator and repress transcription.

Answer: (A) ← Go back to Q**25**

Q26.

Solution

Concept — RNA Splicing: Removal of Introns by the Spliceosome
Eukaryotic pre-mRNA (hnRNA) contains both exons (expressed sequences, retained in mature mRNA) and introns (intervening sequences, removed before translation). After transcription by RNA Pol II, the pre-mRNA undergoes three processing steps: (1) 5' capping (7-methylguanosine cap), (2) 3' polyadenylation (poly-A tail), and (3) **splicing** (intron removal and exon ligation). Splicing is performed by the **spliceosome**, a large ribonucleoprotein complex of five snRNPs (U1, U2, U4, U5, U6) plus numerous accessory proteins.

Step 1 — Mechanism of Splicing:
(i) U1 snRNP base-pairs with the 5' splice site; U2 snRNP binds the branch point adenosine. (ii) The 2'-OH of the branch-point A attacks the 5' splice site (first transesterification), forming a lariat intermediate. (iii) The 3'-OH of the freed upstream exon attacks the 3' splice site (second transesterification), joining the exons and releasing the intron lariat. (iv) The lariat intron is debranched and degraded.

Step 2 — Product:
The mature mRNA (5' cap + coding exons + poly-A tail) is exported to the cytoplasm via nuclear pores and translated by ribosomes. Alternative splicing allows one pre-mRNA to produce multiple protein isoforms.

Why other options are wrong:
Option A: Introns are degraded in the nucleus after removal; they are NOT exported to the cytoplasm or translated. Intron RNA is not a template for protein synthesis.
Option C: In eukaryotes, transcription (in the nucleus) and translation (in the cytoplasm) are spatially separated and temporally uncoupled (pre-mRNA must be processed and exported before translation). Coupled transcription–translation (like in bacteria) does NOT occur in eukaryotes.
Option D: Restriction endonucleases are bacterial enzymes (part of the restriction-modification system) that cut double-stranded DNA at specific palindromic sequences; they have no role in removing RNA introns from pre-mRNA.

Answer: (B) ← Go back to Q26

Q27.

Solution

Concept — Photoperiodism and the Critical Night Length in Short-Day Plants
Photoperiodism is the measurement of the relative lengths of day and night (the photoperiod) to regulate seasonal processes such as flowering, dormancy, and leaf senescence. Crucially, it is the **length of the dark period (night)** that is the decisive signal, not the light period — a fact discovered by Hamner and Bonner (1938) with flash-interruption experiments.

Step 1 — Short-Day Plants (SDPs):
SDPs (e.g. *Xanthium* cocklebur, chrysanthemum, rice, soybean) flower when the uninterrupted dark period **exceeds** a critical minimum length (the "critical night length"). A brief flash of red light (far-red reversible; mediated by phytochrome) interrupting the dark period **resets** the dark-period timer and prevents flowering. This demonstrates that SDPs are really "long-night plants."

Step 2 — Long-Day Plants (LDPs):
LDPs (e.g. spinach, barley, wheat under long days) flower when the dark period is **shorter** than their critical night length (equivalently, when the day is longer than a critical light period). Interrupting the night does NOT prevent their flowering (it can even promote it in LDPs).

Why other options are wrong:
Option A: SDPs require the *dark* period (not the light period) to exceed a critical minimum. They are truly "long-night plants." A long light period would actually prevent flowering in SDPs.
Option B: LDPs flower when the night period is *shorter* than a critical maximum — i.e., when days are long. LDPs do NOT require night length to exceed a maximum.
Option D: Day-neutral plants (e.g. tomato, cucumber, sunflower) flower regardless

of the photoperiod — they do not need a specific day:night ratio and flower under a broad range of conditions.

Answer: (C) ← Go back to Q27

Q28.

Solution

Concept — Abscisic Acid (ABA) and Stomatal Closure: Ion Efflux Mechanism
Abscisic acid (ABA) is synthesised in response to water deficit (drought), high temperature, and salinity. ABA is perceived by its receptors (PYR/PYL/RCAR) in guard cells, triggering a signalling cascade that leads to stomatal closure by reducing guard cell turgor pressure.

Step 1 — Molecular Mechanism of ABA-Induced Closure:
(1) ABA binds PYR/RCAR receptors → (2) inhibition of PP2C phosphatases (ABI1/ABI2) → (3) activation of SnRK2 kinases → (4) phosphorylation and activation of **SLAC1** (slow anion channel 1) and **KAT1** inhibition → (5) efflux of $\mathbf{Cl^-}$ (via SLAC1) and $\mathbf{K^+}$ (via GORK outward K^+ channel) → (6) decreased guard cell osmolarity → (7) water exits guard cells by osmosis → (8) turgor pressure drops → (9) stomata close. ABA also elevates cytosolic Ca^{2+} and produces ROS (H_2O_2), which activate the anion channels.

Step 2 — Stomatal Opening (Contrast):
Stomata open in the morning when blue light activates the H^+-ATPase pump in guard cells, acidifying the apoplast and hyperpolarising the membrane. This activates inward K^+ channels (KAT1) allowing K^+ influx, driving water uptake by osmosis and increasing turgor to open stomata.

Why other options are wrong:
Option A: K^+ *influx* into guard cells causes them to *swell* (open stomata), the exact opposite of what ABA does. ABA promotes K^+ *efflux*.
Option B: ABA does not activate aquaporins to pump water *into* guard cells. Aquaporins are bidirectional water channels; water exits guard cells passively when ion concentration drops (as caused by ABA).
Option C: Callose deposition is a response to wounding and pathogen attack, not a mechanism of stomatal closure. There is no evidence that ABA induces callose deposition to physically block the stomatal pore.

Answer: (D) ← Go back to Q28

Q29.

Solution

Concept — CAM Photosynthesis: Temporal Separation of CO_2 Fixation

CAM (Crassulacean Acid Metabolism) is a photosynthetic strategy used by succulent plants in arid environments (cacti, agave, pineapple, stonecrop). It separates carbon fixation **temporally** (day vs. night) rather than spatially (as in C4 plants). This adaptation reduces water loss by keeping stomata closed during the hot, dry daytime.

Step 1 — Night Phase (CO_2 Fixation):

Stomata *open* at night (cooler, less evaporation). **PEP carboxylase** (not RuBisCO) fixes CO_2: PEP + CO_2 $\rightarrow$ oxaloacetate (OAA) $\rightarrow$ **malate**. Malate is stored as malic acid in the vacuoles. PEP carboxylase has a very high affinity for CO_2 and is not inhibited by O_2 (no photorespiration), making it ideal for nocturnal CO_2 fixation.

Step 2 — Day Phase (Calvin Cycle):

Stomata *close* during the day (conserving water). Stored malate exits vacuoles and is **decarboxylated** by malic enzyme or PEP carboxykinase, releasing CO_2 into the leaf interior. This CO_2 is then fixed by **RuBisCO** into the Calvin cycle in the light (using ATP and NADPH from light reactions). The CO_2 concentration around RuBisCO is high enough to suppress photorespiration even with closed stomata.

Why other options are wrong:

Option B: RuBisCO does not "work more efficiently at cooler temperatures" in any operationally significant way for CAM; it is temperature-sensitive but the primary reason for nocturnal stomatal opening is CO_2 capture by PEP carboxylase and water conservation, not RuBisCO efficiency.

Option C: The light reactions (electron transport, photosystems I and II) require *light* (photons) and occur during the day; they absolutely cannot occur at night. CO_2 fixation at night in CAM uses PEP carboxylase, not RuBisCO.

Option D: CAM is distinct from C4 photosynthesis. C4 plants (maize, sugarcane) have *spatial* separation (mesophyll cells for initial CO_2 fixation by PEP carboxylase; bundle sheath cells for Calvin cycle by RuBisCO) but open their stomata during the day. CAM plants have *temporal* separation within the same cell; they do not require specialised bundle sheath cells.

Answer: (A) ← Go back to Q29

Q30.

Solution

Concept — First Stable Product of CO_2 Fixation in C3 Photosynthesis: 3-PGA

In the Calvin cycle (C3 carbon fixation), CO_2 is combined with ribulose-1,5-bisphosphate (RuBP, a 5-carbon compound) by the enzyme **RuBisCO** (ribulose-1,5-bisphosphate carboxylase/oxygenase). The immediate product is an un-

stable 6-carbon intermediate that rapidly splits into **two molecules of 3-phosphoglycerate (3-PGA)**, each a 3-carbon compound. 3-PGA is therefore the first *stable* product of CO_2 fixation in C3 plants (it was identified by Calvin and Benson using $^{14}CO_2$ and paper chromatography in the 1950s).

Step 1 — Subsequent Steps:
3-PGA is phosphorylated by ATP to 1,3-bisphosphoglycerate (1,3-BPG), then reduced by NADPH to glyceraldehyde-3-phosphate (G3P). G3P is the organic product used for glucose synthesis and for regenerating RuBP. For every 3 CO_2 fixed: 6 molecules of 3-PGA $\rightarrow$ 6 G3P; 5 G3P regenerate 3 RuBP; 1 G3P is net output.

Why other options are wrong:
Option A: Oxaloacetate (OAA) is the first product of CO_2 fixation in **C4 plants** (e.g. maize, sugarcane), where PEP carboxylase adds CO_2 to PEP (3C) to form OAA (4C) in mesophyll cells. In C3 plants, OAA is not involved in initial CO_2 fixation.
Option C: Pyruvate is a 3-carbon compound produced at the *end* of glycolysis (from PEP via pyruvate kinase). It is a product of carbohydrate catabolism, not of photosynthetic CO_2 fixation.
Option D: PEP (phosphoenolpyruvate) is the CO_2 *acceptor* (not the product) in **C4 plants**. In C3 plants, RuBP is the CO_2 acceptor and 3-PGA is the product. PEP is not the first product of the Calvin cycle.

Answer: (B) ← Go back to Q30

Q31.

Solution

Concept — Spermatogenesis: Stages and Spermiogenesis
Spermatogenesis occurs in the seminiferous tubules of the testis and proceeds as follows: (1) **Spermatogonia** (2n) — stem cells that undergo mitosis to produce primary spermatocytes. (2) **Primary spermatocytes** (2n) — undergo meiosis I to form two secondary spermatocytes. (3) **Secondary spermatocytes** (n) — each undergoes meiosis II to form two spermatids. (4) **Spermatids** (n, round cells) — undergo **spermiogenesis** to become spermatozoa. (5) **Spermatozoa** (n, mature motile sperm).

Step 1 — Spermiogenesis in Detail:
Spermiogenesis is a **differentiation** process (NOT a division) in which round spermatids remodel dramatically: (a) the nucleus condenses (chromatin compacted by protamines); (b) the Golgi apparatus produces the **acrosome** (lysosome-like vesicle capping the nucleus, containing hydrolytic enzymes for egg penetration); (c) the **flagellum** develops from the centriole; (d) the **midpiece** forms with a sheath of mitochondria around the axonemal microtubule doublets (9+2 axoneme), pro-

viding ATP for motility; (e) excess cytoplasm is shed as a residual body, phagocytosed by Sertoli cells.

Why other options are wrong:

Option A: Spermatids do NOT undergo any further mitotic division. They are post-meiotic, haploid cells (n). A mitotic division would double the cell number but would not change their haploid status; this is not what occurs. Spermiogenesis is morphological, not proliferative.

Option B: DNA replication is complete before meiosis I in primary spermatocytes. Spermatids are post-meiotic cells with a haploid, already-replicated-and-segregated genome; no further DNA replication occurs.

Option D: Spermiogenesis is an intrinsic differentiation programme supported by Sertoli cells (which provide nutrients and signals); it does not require fusion with a secondary oocyte. Fertilisation of the egg is a separate, subsequent event.

Answer: (C) ← Go back to Q31

Q32.

Solution

Concept — Blastocyst Implantation: Role of the Trophoblast

After fertilisation in the fallopian tube, the zygote undergoes cleavage (morula) and then forms a blastocyst (≈100 cells) by days 4–5. The blastocyst has two distinct regions: (1) the **inner cell mass (ICM / embryoblast)** — gives rise to the embryo proper (all three germ layers); (2) the **trophoblast** — surrounds the blastocoel and is the first cell type to differentiate; it mediates implantation and forms the placenta.

Step 1 — Trophoblast Invasion (Implantation):

By days 6–7, the trophoblast differentiates into: (a) **cytotrophoblast** (inner mononuclear layer) and (b) **syncytiotrophoblast** (outer multinucleated layer formed by cell fusion). The **syncytiotrophoblast** is invasive: it secretes matrix metalloproteinases (MMPs) and other proteolytic enzymes that digest the decidual endometrial stroma, allowing the blastocyst to embed into the endometrium. The syncytiotrophoblast also produces **human chorionic gonadotropin (hCG)**, which maintains the corpus luteum and thus progesterone production to sustain the decidua.

Why other options are wrong:

Option A: The ICM (embryoblast) becomes the embryo, not the invading layer. It is surrounded by the trophoblast. The ICM does not secrete proteases or invade the endometrium.

Option B: Progesterone during implantation is produced by the **ovarian corpus luteum** (maintained by hCG from the syncytiotrophoblast), not the anterior pi-

tuitary. Moreover, progesterone prepares the endometrium for implantation but does not cause the endometrium to engulf the blastocyst.
Option C: The blastocyst does NOT merely adhere to the surface without invasion. Successful implantation requires proteolytic invasion by the syncytiotrophoblast; failure of invasion leads to implantation failure or ectopic pregnancy.

Answer: (D) ← Go back to Q32

Q33.

Solution

Concept — Apomixis and Parthenocarpy: Asexual Reproduction in Plants
Normal angiosperm reproduction involves double fertilisation: (1) one sperm fertilises the egg cell → zygote → embryo; (2) another sperm fuses with the two polar nuclei → triploid endosperm. **Apomixis** and **parthenocarpy** are alternative pathways that bypass fertilisation.

Step 1 — Apomixis:
Apomixis is the asexual production of seeds without fertilisation. The embryo develops from an unfertilised egg (parthenogenesis), from a somatic cell of the ovule (sporophytic apomixis), or from an unreduced embryo sac. Examples: dandelion (*Taraxacum*), hawkweed (*Hieracium*), citrus (nucellar polyembryony). Because no fertilisation occurs, offspring are genetically identical to the mother (clonal). Apomixis is commercially valuable in crop breeding to fix hybrid vigour.

Step 2 — Parthenocarpy:
Parthenocarpy is fruit development without fertilisation (and hence without seed formation or with seedless fruits). Examples: banana, seedless grapes (Thompson), seedless watermelon, cucumber. It can be induced in some crops by applying auxins (IAA, NAA) or gibberellins. Parthenocarpic fruits are diploid (2n) in most natural cases because the fruit wall (pericarp) develops from diploid ovarian tissue.

Why other options are wrong:
Option B: Apomixis does not universally require pollination. In most forms (especially sporophytic/diplospory), neither pollination nor fertilisation is needed. Parthenocarpy also does not require fertilisation; by definition, it bypasses it.
Option C: Both apomixis and parthenocarpy specifically *bypass* double fertilisation — neither is triggered by double fertilisation. If double fertilisation occurred, it would be normal sexual reproduction (or would produce a normal seeded fruit).
Option D: Apomixis can produce seeds via different mechanisms: some produce diploid embryos (sporophytic apomixis, unreduced embryo sacs) while others produce haploid embryos (reduced parthenogenesis). Parthenocarpy does not consistently produce haploid fruits; pericarp tissue is typically diploid regardless.

Answer: (A) ← Go back to Q33

Q34.

Solution

Concept — Industrial Melanism: Natural Selection in Action

Industrial melanism in the peppered moth (*Biston betularia*) is one of the most celebrated real-time demonstrations of natural selection. Before the Industrial Revolution (≈1850s), pale (typical) forms of the moth were common in England; the rare dark (melanic, *carbonaria*) form was quickly eaten by birds on the lichen-covered, pale tree bark. After industrialisation, soot killed the lichen and blackened tree bark in urban areas; dark moths became camouflaged while pale moths became conspicuous.

Step 1 — Natural Selection Mechanism:

H.B.D. Kettlewell (1950s) demonstrated by field experiments (mark-recapture and direct predation observation) that: in polluted industrial woods, dark moths survived at significantly higher rates than pale moths (camouflage advantage); in unpolluted rural woods, the reverse was true. Over decades, the frequency of the dark allele (*carbonaria*) increased dramatically in industrial populations (e.g. in Manchester, >90% dark by 1900 vs. <1% in 1850) — a measurable change in allele frequency due to differential survival (= natural selection by bird predation).

Step 2 — Genetic Basis:

The dark form is controlled by a single dominant allele at the *cortex* gene (a Zn-finger transcription factor locus on chromosome 17). The mutation pre-existed in low frequency before industrialisation; pollution did not create it — selection changed its frequency.

Why other options are wrong:

Option A: The dark colour was NOT directly produced (induced) by industrial pollution or UV radiation. This would be a Lamarckian mechanism (inheritance of acquired characteristics), which is incorrect. The dark allele *pre-existed* as a rare variant and was selectively favoured after pollution changed the environment.

Option C: Dark moths are at a *disadvantage* in clean, non-industrial areas (as Kettlewell showed). There is no universal metabolic superiority; fitness is always environment-dependent.

Option D: Moths do not "voluntarily" migrate based on colour preference or environmental quality. Behaviour is not the mechanism here; differential survival due to predation (differential fitness) is the mechanism of natural selection.

Answer: (B) ← Go back to Q34

Q35.

Solution

Concept — Habitat vs. Ecological Niche (Hutchinson's Hypervolume)

Habitat is the physical place or environment where an organism lives — its "address" (e.g. tropical forest, coral reef, freshwater pond). It describes location.

Ecological niche is the functional role of a species in its ecosystem — its "profession." G.E. Hutchinson (1957) formalised the niche as an n-dimensional hypervolume where each dimension is an abiotic or biotic variable (temperature, humidity, food type, food size, competitor density, parasite load...) within which the species can maintain a viable population. Two species that co-occur in the same habitat almost always have different niches (slight differences in diet, microhabitat, activity time, etc.).

Step 1 — Gause's Competitive Exclusion Principle:

No two species can occupy the same ecological niche (in the same location at the same time) indefinitely. If they try, one will competitively exclude the other. Gause demonstrated this with *Paramecium aurelia* and *P. caudatum* in culture: when grown together with the same resource, one excluded the other. Species that appear to share a habitat survive together by occupying slightly different niches (resource partitioning).

Why other options are wrong:

Option A: This has the definitions *reversed*. Habitat = physical location ("address"); niche = functional role ("profession"). This is a common exam trick reversal.

Option B: Two species can share the same habitat (e.g. multiple bird species in the same forest) but they will always differ in their niches (different prey, different foraging heights, different times). Permanent co-occupancy of the *identical* niche is impossible (competitive exclusion).

Option D: Niche and habitat are distinct concepts with different definitions; using them interchangeably leads to fundamental misunderstandings in ecology.

Answer: (C) ← Go back to Q35

Q36.

Solution

Concept — Cultural Eutrophication: Sequence of Events Leading to Hypoxia

Eutrophication is the enrichment of a water body with nutrients (primarily nitrogen compounds and phosphates) that leads to excessive algal and plant growth. "Cultural" (anthropogenic) eutrophication is accelerated by human inputs: agricultural run-off (fertilisers rich in NO_3^- and PO_4^{3-}), sewage discharge, animal waste, and detergents.

Step 1 — Chain of Events:

(1) **Nutrient enrichment** (N & P input) → (2) **Algal bloom** (explosive growth of cyanobacteria and algae, forming scums) → (3) Algae die (limited by nutrients, light penetration, or seasonal change) → (4) **Microbial decomposition** of dead organic matter by aerobic bacteria (heterotrophs) consumes dissolved O_2 at an enormous rate → (5) **O_2 depletion (hypoxia/anoxia)** in the water column → (6) **Fish kill** (suffocation of fish and other aquatic organisms) and creation of a "dead zone" (e.g. Gulf of Mexico dead zone from Mississippi run-off).

Step 2 — Algal Bloom also Blocks Light:
Dense algal blooms block sunlight penetration, killing submerged aquatic macrophytes that also contribute O_2 to the water. The combined effect of O_2 consumption by decomposers and loss of subaquatic plant O_2 production accelerates hypoxia.

Why other options are wrong:
Option A: This sequence is completely reversed. Eutrophication begins with nutrient enrichment, not with "increased dissolved O_2". Increased O_2 does not initiate the process.
Option B: While algal photosynthesis briefly increases O_2 during daytime, the net long-term effect of eutrophication is O_2 *depletion* (due to decomposition), not increase. Increased fish growth does not result from eutrophication.
Option C: While algae do produce O_2 via photosynthesis, this effect is transient. After algae die, their mass decomposition by bacteria consumes far more O_2 than was produced, resulting in net hypoxia. Fish death, not proliferation, is the final result.

Answer: (D) ← Go back to Q36

Q37.

Solution

Concept — In-situ vs. Ex-situ Conservation of Biodiversity
In-situ conservation ("on-site") protects species within their natural habitats. It addresses the root cause of extinction by protecting and restoring ecosystems. Forms include: **national parks** (strictest protection; no human habitation or resource extraction), **wildlife sanctuaries** (some human activities permitted), **biosphere reserves** (core zone + buffer zone + transition zone; support human communities), marine protected areas, and community-owned reserves. In India: Project Tiger (1973), Project Elephant, Tiger Reserves, and national parks (e.g. Kaziranga, Corbett, Gir) are in-situ efforts.

Step 1 — Advantages of In-situ Conservation:
Species maintain natural behaviours, interactions, and evolutionary potential. Entire ecosystems and ecological processes (pollination, predator–prey dynamics)

are preserved simultaneously. Large areas can protect many species at once (umbrella species concept).

Step 2 — Ex-situ Conservation (Contrast):
Ex-situ conservation protects species outside their natural habitat: (a) zoos (captive animals), (b) botanical gardens (live plants), (c) seed banks (e.g. National Bureau of Plant Genetic Resources, India; Svalbard Global Seed Vault), (d) cryopreservation (gametes, embryos, tissue cultures at $-196°$C), (e) captive breeding programmes with reintroduction goals.

Why other options are wrong:
Option B: Seed banks maintain seeds in temperature-controlled ex-situ facilities. The seeds are removed from their natural habitat and stored artificially — this is ex-situ conservation.
Option C: Captive breeding programmes for tigers in zoological parks are ex-situ conservation. The animals are kept outside their natural habitat; even if reintroduction is the goal, the breeding itself is ex-situ.
Option D: Cryopreservation of gametes, embryos, or tissues in liquid nitrogen is a form of ex-situ conservation at the gametic/cellular level. It is valuable for rare species but is entirely off-site.

Answer: (A) ← Go back to Q37

Q38.

Solution

Concept — Secondary Succession vs. Primary Succession
Ecological succession is the directional, predictable change in species composition and community structure over time. **Primary succession** begins on bare, sterile substrate with no pre-existing soil (e.g. newly formed volcanic lava flow, retreating glacier, freshly exposed rock). It is extremely slow (centuries to millennia) because the pioneer organisms (lichens, then mosses) must build soil from scratch. **Secondary succession** begins on substrate that has been disturbed but retains its soil, organic matter, seed bank, and spore bank from the previous community.

Step 1 — Why Secondary Succession is Faster:
The pre-existing soil provides: (a) mineral nutrients, (b) organic humus for water retention and microbial activity, (c) dormant seeds (seed bank) and vegetative structures (roots, rhizomes), (d) soil microbiota (mycorrhizal fungi, nitrogen-fixing bacteria). The successional sequence in secondary succession starts at a more advanced stage (often directly to herbaceous plants or shrubs), skipping the pioneer lichen/moss stages.

Step 2 — Examples of Secondary Succession:
Abandoned agricultural field (old field succession) $\rightarrow$ annual weeds $\rightarrow$ perennial

grasses → shrubs → early-succession trees → late-succession (climax) forest. Recovery after a forest fire (if soil is intact), or after a flood. Timescale: decades to a few centuries (much faster than primary succession).

Why other options are wrong:
Option A: Secondary succession occurs in both terrestrial (abandoned fields, logged forests, fire sites) and aquatic (recovering ponds after drought) habitats. It is not restricted to aquatic environments.
Option C: Volcanic eruptions with complete lava flows that destroy soil and life initiate *primary* succession (on fresh lava), not secondary. Secondary succession requires *intact soil* to remain after the disturbance.
Option D: The climax community is determined by regional climate ("climatic climax"): tropical rainforest in humid tropics, temperate deciduous forest in temperate climates, taiga (boreal forest) in subarctic regions, grassland in semi-arid regions. Secondary succession does NOT always lead to a grassland; the climax reflects the regional climate.

Answer: (B) ← Go back to Q38

Q39.

Solution

Concept — ELISA (Enzyme-Linked Immunosorbent Assay): Positive Result Indicator
ELISA is a plate-based immunoassay that uses enzyme-linked antibodies to detect and quantify antigens or antibodies. In a **sandwich ELISA** for HIV diagnosis (detecting anti-HIV antibodies in patient serum): (1) HIV antigens (e.g. gp120, p24) are coated on the well. (2) Patient serum is added; if anti-HIV antibodies are present, they bind the immobilised antigen. (3) An enzyme-linked anti-human IgG secondary antibody is added; it binds the captured anti-HIV antibodies. (4) A chromogenic substrate (e.g. TMB = 3,3',5,5'-tetramethylbenzidine for HRP; pNPP for alkaline phosphatase) is added. (5) The enzyme cleaves the substrate → colour change.

Step 1 — Positive Result:
A **colour change** (yellow/blue for TMB with HRP) measured spectrophotometrically (OD_{450}) **above the cut-off absorbance** indicates a POSITIVE result — i.e. anti-HIV antibodies are present in the sample. The intensity is proportional to antibody concentration.

Step 2 — Negative Result:
If no anti-HIV antibodies are present in the serum, the secondary enzyme-linked antibody does not bind and is washed away ⇒ substrate is not cleaved ⇒ **no colour change** = negative result.

Why other options are wrong:
Option A: Absence of colour change indicates a *negative* result (no anti-HIV antibodies detected), not a positive result. This is the opposite of the correct answer.
Option B: Agglutination of red blood cells is a characteristic of different immunological tests, such as the Coombs test, blood typing (ABO/Rh hemagglutination assay), and the Widal test (for typhoid). ELISA does not use RBCs or detect agglutination.
Option D: Precipitin line formation in agarose gel describes **immunodiffusion** techniques (Ouchterlony double diffusion, immunoelectrophoresis). ELISA is a plate-based, colorimetric method; it does not use agarose gel or detect precipitin lines.

Answer: (C) ← Go back to Q**39**

Q40.

Solution

Concept — RNA Interference (RNAi): Post-Transcriptional Gene Silencing via RISC
RNAi is a conserved eukaryotic pathway triggered by double-stranded RNA (dsRNA). It was discovered by Fire and Mello in *C. elegans* (1998, Nobel Prize 2006). The pathway: (1) Long dsRNA (from viral replication, transposons, or experimental introduction) is cleaved by the enzyme **Dicer** (a type III RNase) into short dsRNA duplexes of 21–23 bp called **siRNA** (small interfering RNA). (2) siRNA is loaded into the **RISC** (RNA-Induced Silencing Complex), a multi-protein complex containing **Argonaute** (AGO2) as the catalytic component. (3) Within RISC, the sense (passenger) strand of siRNA is degraded; the antisense (**guide**) strand remains associated with AGO2. (4) The guide strand directs RISC to complementary mRNA sequences. (5) AGO2's Slicer activity **cleaves the target mRNA** between positions 10 and 11 of the siRNA. (6) The cleaved mRNA is degraded by cellular exonucleases. Result: **gene silencing at the post-transcriptional level**.

Step 1 — Applications:
RNAi is used in agriculture (e.g. silencing insect digestive genes for pest resistance in Bt-cotton enhancement; gene silencing in *Agrobacterium*-transformed plants to reduce enzyme browning). In medicine: experimental siRNA drugs (e.g. patisiran, the first siRNA drug for TTR amyloidosis, FDA 2018) deliver siRNA to silence disease-causing genes.

Why other options are wrong:
Option A: RNAi acts *post-transcriptionally* — it targets mRNA for degradation, not RNA polymerase at the promoter. Blocking RNA polymerase at the promoter would be *transcriptional* silencing (a different mechanism, e.g. promoter methyla-

tion or transcription factor competition). RNAi does not affect the gene's DNA or its transcription directly.
Option B: CpG promoter methylation leading to heterochromatin is *epigenetic transcriptional silencing* — a fundamentally different, often permanent mechanism from RNAi. RNAi-directed DNA methylation (RdDM, RNA-directed DNA methylation) does exist in plants but is distinct from the canonical siRNA/RISC mRNA cleavage described as RNAi.
Option C: siRNA does NOT incorporate into the ribosome. siRNA is incorporated into **RISC** (a cytoplasmic silencing complex), not into ribosomes (which translate mRNA into protein). Ribosomes contain rRNA (18S, 28S, etc.) and ribosomal proteins; siRNA is not a ribosomal component.

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