BITSAT Biology Sample Paper – 5

Duration: 60 Minutes **Maximum Marks: 120**

Instructions

- This paper contains **40** Multiple Choice Questions (Single Correct Answer).
- Each correct answer carries **+3 marks**. Each incorrect answer carries **−1** mark. Unattempted questions carry **0** marks.
- Only **one** option is correct. Choose carefully.
- Use of mobile phones, calculators, or electronic gadgets is strictly prohibited.

Q1. In plant cells, cytokinesis (cytoplasmic division at the end of mitosis) differs fundamentally from animal cytokinesis in that plant cells:

(A) Use a contractile actin-myosin ring to pinch the cell in two

(B) Form a new cell plate at the centre of the dividing cell, built from Golgi-derived vesicles fusing along the phragmoplast (a complex of microtubules between the daughter nuclei); the plate grows outward until it fuses with the existing cell wall

(C) Do not undergo cytokinesis at all

(D) Use proteasome-mediated degradation of the parent cell

Q2. Sphingolipids are membrane lipids based on the long-chain amino-alcohol **sphingosine**, rather than glycerol. Their key biological role includes:

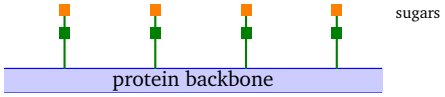
(A) Acting as the primary energy storage molecule in adipose tissue

(B) Forming the inner mitochondrial membrane

(C) Serving as major components of neuronal myelin sheaths (especially sphingomyelin, ceramides, gangliosides) and concentrating in lipid rafts; defects cause lysosomal storage diseases (Tay-Sachs, Gaucher, Niemann-Pick)

(D) Acting as enzymes (catalysts) themselves

Q3. Glycoproteins are proteins covalently bound to oligosaccharide chains, as shown below. The sugar chains are added in:



(A) The cytosol on free ribosomes

(B) The nucleus

(C) The mitochondrial matrix

(D) The lumen of the endoplasmic reticulum (*N*-linked, on asparagine residues; transferred en bloc from dolichol-phosphate) and modified further in the Golgi apparatus (where *O*-linked glycosylation on serine/threonine also occurs)

Q4. Gluconeogenesis — the synthesis of glucose from non-carbohydrate precursors (lactate, glycerol, glucogenic amino acids) — is the metabolic mirror of glycolysis. It occurs predominantly in:

(A) The cytosol of liver hepatocytes (and to a smaller extent kidney cortex), and is essential during fasting to maintain blood glucose; brain and red blood cells depend obligately on circulating glucose

(B) The mitochondrial matrix of muscle cells

(C) The nucleus of all cells

(D) Adipose tissue exclusively

Q5. In the absence of oxygen, mammalian skeletal muscle cells regenerate NAD^+ for continued glycolysis by:

(A) Producing ethanol and CO_2 (this is yeast alcoholic fermentation, not animal)

(B) Donating electrons directly to oxygen

(C) Reducing pyruvate to lactate using NADH, catalysed by lactate dehydrogenase (LDH); the lactate is exported to the blood, taken up by the liver, and reconverted to glucose via the **Cori cycle**

(D) Releasing acetyl-CoA into the cytosol

Q6. The enormous absorptive surface area of the small intestine ($\sim 200\,\mathrm{m}^2$ in adults — comparable to a tennis court) is achieved by:

(A) Simple flat epithelium with no folds

(B) Only macroscopic folds visible to the eye

(C) A single layer of squamous cells

(D) Three levels of folding: **plicae circulares** (visible large folds, $\sim 3\times$); **villi** (finger-like projections of mucosa, $\sim 10\times$); **microvilli** (cell-surface projections of enterocytes forming the brush border, $\sim 20\times$)

Q7. Adult human haemoglobin (HbA) has the quaternary structure:

(A) Tetrameric — two α and two β globin chains ($\alpha_2\beta_2$), each carrying one haem (iron-protoporphyrin IX) prosthetic group, so each Hb tetramer binds up to four O_2 molecules

(B) Monomeric — a single polypeptide

(C) Dimeric — two identical chains

(D) Eight subunits ($\alpha_4\beta_4$)

Q8. The Frank-Starling law of the heart states that:

(A) Heart rate is independent of venous return

(B) Within physiological limits, the heart pumps out (stroke volume) whatever blood is delivered to it (venous return); greater end-diastolic stretch of cardiac myofibres $\rightarrow$ stronger contraction (optimum sarcomere length $\sim 2.2\,\mu\mathrm{m}$); this matches output of the two ventricles automatically

(C) Cardiac output is fixed regardless of activity

(D) Heart muscle does not respond to stretch

Q9. The medullary concentration gradient that allows mammalian kidneys to produce concentrated urine (osmolarity up to $1200\,\mathrm{mOsm}$ in humans, far higher in desert mammals) is established and maintained by:

(A) Active secretion of NaCl in the proximal tubule

(B) Passive diffusion of glucose into the medulla

(C) ADH alone, in the absence of any anatomical specialisation

(D) The **counter-current multiplier** action of the loop of Henle (descending limb water-permeable, ascending limb actively pumps NaCl) plus the counter-current exchange in the vasa recta capillaries that preserve the gradient

Q10. At the vertebrate neuromuscular junction (NMJ) between a motor neuron and a skeletal muscle fibre, the neurotransmitter is acetylcholine (ACh), which binds receptors on the muscle membrane that are:

(A) Nicotinic acetylcholine receptors — pentameric ligand-gated cation channels; binding of two ACh molecules opens the channel, allowing Na^+ influx, triggering the end-plate potential and ultimately muscle action potential and contraction

(B) G-protein-coupled muscarinic receptors (these are at parasympathetic effector organs, not NMJ)

(C) Adrenergic receptors

(D) $GABA_A$ receptors

Q11. Parathyroid hormone (PTH), secreted by the four parathyroid glands embedded in the posterior thyroid, is the master regulator of plasma calcium. In response to LOW blood Ca^{2+}, PTH:

(A) Lowers blood calcium by inhibiting bone resorption

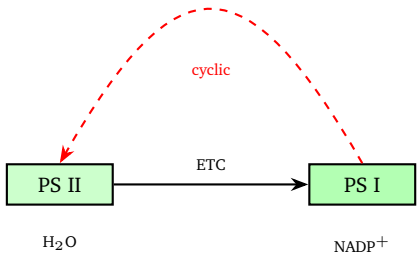
(B) Raises blood calcium by (i) stimulating osteoclast-mediated bone resorption, (ii) increasing Ca^{2+} reabsorption in the kidney DCT (and

decreasing phosphate reabsorption), (iii) activating 1α-hydroxylation of vitamin D to calcitriol, which boosts intestinal Ca^{2+} absorption

(C) Acts directly on the gut to absorb calcium (this is calcitriol's role, not PTH directly)

(D) Has no effect on bone

Q12. The Z-scheme of photosynthesis, shown below, alternates between linear (non-cyclic) and cyclic electron flow. In cyclic photophosphorylation:



(A) Both PS I and PS II participate, and water is split

(B) NADPH is generated as the main product

(C) Only PS I operates; electrons cycle from PS I through the cytochrome b_6f complex back to PS I, generating ATP only (no NADPH, no O_2); important when ATP demand exceeds NADPH demand

(D) No light is required

Q13. In Mendelian genetics, when observed offspring numbers deviate from expected Mendelian ratios, statistical analysis is performed using the:

(A) Chi-square (χ^2) goodness-of-fit test: $\chi^2 = \sum(O - E)^2/E$, where O is the observed count and E the expected count; compared against χ^2 critical values at the appropriate degrees of freedom (number of classes minus 1) and significance level ($p = 0.05$)

(B) ANOVA

(C) Student's t-test

(D) Pearson correlation

Q14. Human skin colour, height, and intelligence are examples of *polygenic* (or quantitative) traits because they:

(A) Are controlled by single genes with two alleles

(B) Result from the additive effects of many genes (typically 5–20, each contributing a small effect), often modified by environmental factors; phenotypes show continuous variation (normal distribution) rather than discrete classes; described by Nilsson-Ehle's wheat-kernel-colour experiment (1909)

(C) Are determined entirely by the environment

(D) Follow strict $9:3:3:1$ ratios

Q15. Post-translational modifications (PTMs) such as phosphorylation, acetylation, methylation, glycosylation and ubiquitination expand the functional repertoire of the proteome by:

(A) Altering the DNA sequence

(B) Producing entirely new genes

(C) Reversibly changing protein activity, stability, localisation, interactions, and degradation; e.g. phosphorylation toggles many signalling proteins on and off; histone modifications control chromatin accessibility (the "histone code"); ubiquitination tags proteins for proteasomal destruction

(D) Are limited to bacterial proteins

Q16. The *trp* operon of *E. coli*, which encodes enzymes for tryptophan biosynthesis, is normally:

(A) Inducible by tryptophan (this describes the *lac* operon's behaviour with lactose)

(B) Always expressed regardless of conditions

(C) Activated by tryptophan binding directly to RNA polymerase

(D) Repressible — ON by default; when tryptophan is plentiful, it acts as a co-repressor, binding the trp repressor protein and enabling it to

bind the operator, blocking transcription. When tryptophan is scarce, the repressor is inactive and the operon transcribes (a feedback-repression circuit complemented by attenuation)

Q17. The Miller-Urey experiment (1952), a foundational test of the chemical-evolution hypothesis of life's origin, demonstrated that:

(A) DNA can self-replicate without enzymes

(B) When a flask containing methane, ammonia, hydrogen and water vapour (simulating Oparin-Haldane's reducing primitive atmosphere) was subjected to electric sparks (simulating lightning) for a week, several amino acids (glycine, alanine, aspartic acid) and other simple organic molecules were spontaneously formed

(C) Modern bacteria evolved from yeast

(D) Cells were created in a test tube

Q18. During mammalian fertilisation, the species-specific binding of sperm to the egg occurs via interaction with which egg-surface glycoprotein, located in the zona pellucida?

(A) ZP1 (structural; forms crosslinks but does not bind sperm directly)

(B) ZP2 (acts as a secondary sperm receptor after acrosome reaction)

(C) ZP3 (also called Zona Pellucida glycoprotein 3) — the primary sperm receptor; binding of ZP3 to its complementary protein on the sperm head triggers the acrosome reaction, releasing hydrolytic enzymes (hyaluronidase, acrosin) that allow the sperm to penetrate the zona

(D) Vitelline membrane glycoprotein

Q19. Around the 5th–6th day after fertilisation, the human embryo has developed into a hollow, fluid-filled structure with an inner cell mass and an outer trophoblast layer. This stage is called the:

(A) Zygote (the single-celled fertilised egg)

(B) Morula ($\sim$ 16-cell solid ball, day 3–4)

(C) Gastrula (post-implantation, after the formation of three germ layers)

(D) Blastocyst — the structure that hatches from the zona pellucida and implants in the uterine endometrium around day 6–8; the inner cell mass becomes the embryo proper, the trophoblast becomes the chorion and placenta

Q20. In males, follicle-stimulating hormone (FSH) from the anterior pituitary acts on:

(A) Sertoli cells of the seminiferous tubules, stimulating spermatogenesis, androgen-binding protein (ABP) production, and inhibin secretion (feedback inhibition of FSH)

(B) Leydig cells, stimulating testosterone production (this is the role of LH)

(C) Prostate gland (this is androgen-responsive but not by FSH)

(D) Adrenal cortex

Q21. The combined oral contraceptive pill (containing synthetic oestrogen + progestin) prevents pregnancy primarily by:

(A) Acting as a physical barrier

(B) Killing sperm directly

(C) Suppressing the mid-cycle LH surge (and FSH) via negative feedback on hypothalamus and anterior pituitary, thereby inhibiting ovulation; it also thickens cervical mucus and alters endometrial receptivity — a triple-acting contraceptive

(D) Triggering immediate abortion of any fertilised egg

Q22. Parthenocarpy is the development of fruit:

(A) From an unfertilised endosperm only

(B) With a fully formed seed inside

(C) Only in monocot plants

(D) Without fertilisation of the ovule — so the resulting fruit lacks seeds; examples include banana (cultivated triploid varieties), pineapple, seedless grapes, and many seedless oranges. Can occur naturally or be induced by spraying with auxin/GA_3

Q23. Cytokinins (purine-derived plant hormones, named for their role in promoting cytokinesis) primarily:

(A) Promote cell division (cytokinesis), counteract apical dominance by promoting lateral bud outgrowth, delay leaf senescence ("Richmond-Lang effect"), and are essential for shoot regeneration in tissue culture (high cytokinin/auxin ratio favours shoots; low ratio favours roots)

(B) Cause stem elongation

(C) Promote fruit ripening

(D) Trigger seed dormancy

Q24. Plant roots growing downward in response to gravity, and shoots growing upward, illustrate:

(A) Phototropism (response to light)

(B) Gravitropism (geotropism) — shoots are negatively gravitropic; roots positively gravitropic; mediated by sedimentation of dense starch-filled amyloplasts (statoliths) in specialised cells (columella cells of the root cap, endodermis of shoots), triggering asymmetric auxin redistribution

(C) Thigmotropism (response to touch)

(D) Hydrotropism (response to water)

Q25. Certain filamentous cyanobacteria such as *Nostoc* and *Anabaena* carry out atmospheric N_2 fixation in specialised, thick-walled cells called:

(A) Spores

(B) Bacteroids

(C) Rhizoids

(D) Heterocysts — thick-walled, micro-aerobic (low-O_2) cells that house the oxygen-sensitive nitrogenase enzyme, protecting it from inactivation by photosynthetic O_2 produced in neighbouring vegetative cells. Spaced regularly along the filament (every 10–20 cells)

Q26. A lichen is best described as:

(A) A symbiotic, mutualistic association between a fungus (mycobiont, usually an Ascomycete) and a photosynthetic partner (phycobiont — a green alga and/or cyanobacterium); the fungus provides structure, water and minerals; the alga/cyanobacterium provides photosynthate. Lichens are excellent bio-indicators of air pollution

(B) A parasitic fungus on a tree

(C) A type of bryophyte

(D) A simple seaweed

Q27. Phylum Mollusca (second-largest animal phylum, $\sim 85{,}000$ species: snails, slugs, octopuses, squid, oysters, mussels) is characterised by:

(A) Jointed appendages and chitinous exoskeleton (this is Arthropoda)

(B) A soft, unsegmented body typically divided into head, muscular foot, and visceral mass; covered by a mantle that may secrete a calcareous shell; respiration via gills (ctenidia) or, in pulmonate snails, a vascularised mantle cavity; the radula — a chitinous rasping organ — for feeding

(C) A notochord at some stage

(D) Stinging cells (this is Cnidaria)

Q28. Dengue fever, a re-emerging arboviral disease causing ~ 100–400 million infections annually, is caused by:

(A) *Plasmodium* sporozoites transmitted by *Anopheles* mosquito

(B) Bacterial infection transmitted by tsetse fly

(C) Dengue virus (DENV, a single-stranded RNA Flavivirus with four serotypes 1–4) transmitted by the day-biting female *Aedes aegypti* and *Aedes albopictus* mosquitoes; severe form is dengue haemorrhagic fever / dengue shock syndrome

(D) A spirochaete transmitted by tick

Q29. The Major Histocompatibility Complex (MHC) proteins on cell surfaces are critical to adaptive immunity because they:

(A) Present peptide antigens to T-cell receptors. **MHC class I** (on all nucleated cells) presents endogenous (intracellular) peptides to $CD8^+$ cytotoxic T cells. **MHC class II** (on antigen-presenting cells: dendritic cells, macrophages, B cells) presents exogenous peptides to $CD4^+$ helper T cells. MHC mismatch is the basis of transplant rejection

(B) Are secreted antibodies in serum

(C) Act as complement proteins

(D) Are exclusively on red blood cells

Q30. Opium and its derivatives (morphine, heroin, codeine) are highly addictive drugs of abuse classified as:

(A) CNS stimulants

(B) Opioid CNS depressants — they bind μ, δ and κ opioid receptors (G-protein-coupled receptors of the endogenous endorphin/enkephalin system) producing potent analgesia, sedation, euphoria, respiratory depression and constipation; chronic use causes tolerance, physical dependence and severe withdrawal

(C) Hallucinogens like LSD

(D) Anabolic steroids

Q31. In ecology, *r*-selected species and *K*-selected species represent two extremes of life-history strategy. *r*-selected species typically:

(A) Live long and produce few well-cared-for offspring (this is K-selected)

(B) Reach population sizes far above carrying capacity sustainably

(C) Are characterised by short life span, early maturity, high reproductive rate, many small offspring with little parental care; thrive in disturbed/unstable environments (insects, weeds, rodents, many fish); population size fluctuates dramatically around the environmental carrying capacity (K)

(D) Have stable populations near K (this is K-selected)

Q32. When a community begins to develop on a previously sterile substrate (newly cooled lava flow, bare rock exposed by glacial retreat, a sand dune, an abandoned quarry), the process is termed:

(A) Secondary succession (begins on disturbed but not sterile ground — after fire, agricultural abandonment)

(B) Climax community

(C) Stratification of a stable forest

(D) Primary succession — starts with pioneer species (lichens, cyanobacteria, mosses) that gradually weather the rock and build the first soil; followed by grasses, herbs, shrubs, and eventually forest; takes many centuries to reach climax

Q33. The CRISPR-Cas9 system, awarded the 2020 Chemistry Nobel Prize to Emmanuelle Charpentier and Jennifer Doudna, allows precise genome editing because:

(A) It uses random mutagenesis

(B) A guide RNA (gRNA, $\sim$ 20 nucleotides) base-pairs with the target DNA sequence (next to a PAM site), directing the Cas9 endonuclease to introduce a double-strand break at that exact location; the cell

then repairs the break by non-homologous end joining (NHEJ; produces indels $\rightarrow$ gene knockout) or homology-directed repair (HDR; allows precise insertions)

(C) It removes only single nucleotides at random

(D) It is restricted to plant cells

Q34. Golden Rice (Oryza sativa cv.) is a genetically modified rice variety developed to address childhood blindness and mortality from vitamin A deficiency. It carries:

(A) A gene for Bt insecticidal toxin

(B) A herbicide-resistance gene

(C) Phytoene synthase (*psy*) from daffodil/maize and crtI (carotene desaturase) from *Erwinia* bacterium; these introduce the β-carotene biosynthesis pathway into rice endosperm, giving the grain a yellow-orange colour; consumers convert β-carotene to vitamin A

(D) A flowering-time gene

Q35. Influenza pandemics (such as 1918 Spanish flu, 2009 H1N1 swine flu) arise from the rapid antigenic change of the influenza A virus. The principal mechanisms generating new pandemic strains are:

(A) Bacterial mutation

(B) Insertional mutagenesis only

(C) Recombination between only two human strains

(D) Antigenic drift (gradual point mutations in HA/NA genes), and more dramatically antigenic shift (reassortment of the segmented RNA genome when two influenza strains co-infect one animal — often pigs — creating a novel HA/NA combination to which humans have no prior immunity)

Q36. In an allergic reaction (immediate, type-I hypersensitivity), the molecule released from mast cells and basophils upon allergen-IgE crosslinking,

causing vasodilation, increased capillary permeability and bronchoconstriction, is:

(A) Histamine, released within seconds from preformed granules of mast cells (and basophils); leukotrienes, prostaglandins, cytokines follow later. Antihistamines (cetirizine, loratadine) are H_1-receptor blockers; severe anaphylaxis needs adrenaline (epinephrine)

(B) Insulin

(C) Renin

(D) Thyroxine

Q37. Among the three classical ecological pyramids (numbers, biomass, energy), the pyramid of **energy** is always upright (broad base, narrowing top) because:

(A) The number of organisms always decreases up the food chain

(B) Biomass always increases up the food chain

(C) By Lindemann's $\sim 10\%$ rule, only $\sim 10\%$ of the energy in one trophic level is transferred to the next; the rest is lost as heat (respiration), undigested material, and faeces. Energy flow is unidirectional and continuously dissipated, so the pyramid can never invert

(D) Decomposers add energy back at every level

Q38. The final, relatively stable, self-perpetuating community that develops at the end of ecological succession in a given region is called the:

(A) Sere

(B) Pioneer community

(C) Sub-climax community

(D) Climax community — in equilibrium with the prevailing climate and substrate; composed of dominant species (e.g. oak-hickory deciduous forest in temperate eastern North America; tropical evergreen forest in equatorial regions); shows maximum biodiversity, niche specialisation and stable nutrient cycling

Q39. Gibberellins (GAs) are tetracyclic diterpenoid plant hormones first isolated from the fungal pathogen *Gibberella fujikuroi* (cause of "bakanae" or "foolish seedling" disease of rice). Their physiological roles include:

(A) **Breaking seed and bud dormancy** (counteracting ABA), promoting internode elongation/bolting (especially in rosette plants like cabbage on long days), stimulating α-amylase in barley aleurone (basis of commercial malt production), and promoting parthenocarpic fruit set in seedless grapes

(B) Causing fruit abscission

(C) Triggering stomatal closure

(D) Inhibiting seed germination

Q40. Restriction enzymes (restriction endonucleases) such as *EcoRI* are named by an established convention. The letters in "*EcoRI*" indicate:

(A) Random naming with no significance

(B) "*E*" for *Escherichia*, "*co*" for *coli*, "R" for strain RY13, and "I" for the first such enzyme isolated from that strain. The genus is italicised with a capital first letter; the species name italicised lowercase; strain in roman; Roman numeral for the order of identification

(C) Discovery date

(D) Just the discoverer's initials

Detailed Solutions

Q1.

Solution

Concept — Plant vs animal cytokinesis: The two domains of life solve cytoplasmic division very differently because of the rigid plant cell wall.

Step 1 — Animal cytokinesis (cleavage): A contractile ring of actin filaments and myosin II forms beneath the plasma membrane at the equator. As ATP hydrolysis fuels myosin sliding, the ring tightens like a purse string, pinching the cell into two daughters (cleavage furrow).

Step 2 — Plant cytokinesis (cell-plate formation): The rigid cell wall prevents furrowing. Instead:

- Golgi-derived vesicles (carrying cell-wall precursors and membrane material) are transported to the cell equator along microtubules of the **phragmoplast**.
- Vesicles fuse at the centre to form a flattened, growing **cell plate**.
- Cell plate expands outward (centrifugally) toward the parental cell wall.
- When the cell plate fuses with the lateral cell walls, two daughter cells are formed.
- The plate matures into the middle lamella + two new primary walls + two new plasma membranes.

Step 3 — Phragmoplast components: Two opposed sets of microtubules (with + ends overlapping at the equator), plus actin and many transport motor proteins (kinesins, myosins). Key plant-specific.

Final Answer: Cell plate from Golgi vesicles along phragmoplast $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q1

Q2.

Solution

Concept — Sphingolipids: A major class of membrane lipids built on the long-chain amino-alcohol **sphingosine**, not glycerol. The simplest is ceramide (sphingosine + fatty acid); modifications give the diverse family.

Step 1 — Major sphingolipids:

- **Ceramide:** sphingosine + fatty acid; signalling and apoptosis.

- **Sphingomyelin:** ceramide + phosphocholine head group; major component of myelin sheath insulating nerve axons.
- **Cerebrosides and globosides:** ceramide + neutral sugars; common in nerve cell membranes.
- **Gangliosides:** ceramide + complex oligosaccharides containing sialic acid; abundant in neuronal plasma membranes (especially the outer leaflet), involved in cell-cell recognition.

Step 2 — Lipid rafts: Sphingolipids and cholesterol cluster into thicker, more ordered, less fluid microdomains in cell membranes called lipid rafts; these recruit specific signalling proteins (Src-family kinases, GPI-anchored proteins).

Step 3 — Diseases of sphingolipid metabolism: Lysosomal storage disorders are caused by inherited deficiency of sphingolipid-catabolising enzymes:

- Tay-Sachs disease: hexosaminidase A deficiency $\rightarrow$ GM2 ganglioside accumulates.
- Gaucher disease: glucocerebrosidase deficiency.
- Niemann-Pick disease: sphingomyelinase deficiency.
- Krabbe disease (globoid cell leukodystrophy): galactosylceramidase deficiency.

Final Answer: Myelin component; lipid rafts; storage diseases $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q2

Q3.

Solution

Concept — Glycoproteins: Proteins covalently bound to oligosaccharides. The vast majority of membrane and secretory proteins are glycosylated — an essential post-translational modification.

Step 1 — Two types of glycosylation:

- **N-linked:** sugar chain attached to side-chain nitrogen of an **asparagine** residue (in the consensus sequence N-X-S/T, where X is any aa except proline). Initial 14-sugar oligosaccharide is transferred en bloc from dolichol-phosphate (a long-chain isoprenoid lipid in ER membrane) by oligosaccharyltransferase **in the ER lumen, co-translationally**.
- **O-linked:** sugar attached to side-chain oxygen of a **serine or threonine** residue. Added one sugar at a time, post-translationally, in the Golgi apparatus.

Step 2 — Processing in Golgi: N-linked glycan undergoes extensive trimming and modification as the glycoprotein passes through cis $\rightarrow$ medial $\rightarrow$ trans Golgi cisternae. Sugars added: GlcNAc, Gal, sialic acid (NeuAc), fucose. Result: highly diverse, often branched, mature glycan structures.

Step 3 — Functions of protein glycosylation:

- Protein folding (ER quality control via calnexin/calreticulin chaperone cycle).
- Stability and protease resistance.
- Cell-surface recognition (ABO blood groups, MHC, selectins).
- Targeting (mannose-6-phosphate tag for lysosomal proteins).
- Antibody effector function (Fc region glycosylation tunes ADCC).

Step 4 — Examples of glycoproteins: Immunoglobulins, hormones (FSH, LH, hCG, EPO), cell-adhesion molecules (cadherins, integrins), mucins (very heavily O-glycosylated, $> 50\%$ sugar by mass).

Final Answer: ER (N-linked) and Golgi (O-linked + processing) $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q3

Q4.

Solution

Concept — Gluconeogenesis — making glucose from scratch: Glucose is essential for brain ($\sim 120\,\text{g}\,\text{day}^{-1}$) and RBCs (obligate glucose users). Between meals or during fasting, the liver maintains blood glucose by mobilising glycogen first (glycogenolysis), then by synthesising glucose from non-carbohydrate sources (gluconeogenesis).

Step 1 — Substrates (precursors):

- Lactate from anaerobic glycolysis (muscle, RBC) — the **Cori cycle**.
- Glycerol from triglyceride hydrolysis in adipose tissue.
- Glucogenic amino acids (alanine via the glucose-alanine cycle; aspartate, glutamate, etc.) from muscle protein.
- Propionate (in ruminants from microbial fermentation).

Step 2 — Location and timing:

- Liver hepatocytes (most quantitative).
- Kidney cortex (significant during prolonged fasting and acidosis).

- Occurs in cytosol (most steps) and mitochondrion (pyruvate carboxylase).
- Not in muscle, brain, RBCs (lack glucose-6-phosphatase).

Step 3 — Pathway: Essentially reverse of glycolysis but bypasses three irreversible steps using four unique gluconeogenic enzymes:

(a) Pyruvate carboxylase + PEP carboxykinase (PEPCK): bypass pyruvate kinase.
(b) Fructose-1,6-bisphosphatase: bypass phosphofructokinase-1.
(c) Glucose-6-phosphatase: bypass hexokinase; only in liver and kidney (and intestine) ER lumen.

Net: 6 ATP/GTP consumed per glucose synthesised (much more than the 2 ATP produced by glycolysis), so gluconeogenesis is energetically expensive.

Step 4 — Hormonal control:

- Glucagon, cortisol, adrenaline stimulate gluconeogenesis (fasting state).
- Insulin inhibits gluconeogenesis (fed state).

Final Answer: Cytosol of hepatocytes (and kidney cortex) $\Rightarrow \boxed{\text{A}}$

Answer: (A) Go Back to Q4

Q5.

Solution

Concept — Anaerobic regeneration of NAD^+: Glycolysis produces 2 NADH per glucose. Without oxygen, the mitochondrial ETC cannot oxidise NADH back to NAD^+. To keep glycolysis going (the only ATP source in anaerobic conditions), NAD^+ must be regenerated by a different pathway: **fermentation**.

Step 1 — Lactic acid (homolactic) fermentation: In skeletal muscle during intense exercise, RBCs (which lack mitochondria), and most lactic acid bacteria:

$$\text{Pyruvate} + \text{NADH} \xrightarrow{\text{LDH}} \text{Lactate} + \text{NAD}^+$$

Lactate dehydrogenase (LDH) catalyses the reversible reduction. NAD^+ is recycled. ATP yield remains only 2 per glucose (net), but glycolysis can continue.

Step 2 — The Cori cycle: Lactate from muscle is exported into blood $\rightarrow$ liver. Liver uses gluconeogenesis to reconvert lactate $\rightarrow$ pyruvate $\rightarrow$ glucose, which is exported back to muscle. This shifts the ATP cost (gluconeogenesis is energetically

expensive) from muscle to liver.

Step 3 — Alcoholic fermentation (yeast):

$$\text{Pyruvate} \xrightarrow{\text{PDC}} \text{Acetaldehyde} + CO_2 \xrightarrow{\text{ADH}} \text{Ethanol}$$

Pyruvate decarboxylase removes CO_2; alcohol dehydrogenase reduces acetaldehyde to ethanol, regenerating NAD^+. Basis of brewing (beer, wine, spirits), breadmaking (CO_2 rises dough), bioethanol production.

Step 4 — Lactic acid bacteria in dairy: *Lactobacillus*, *Streptococcus* ferment milk sugar (lactose) $\rightarrow$ lactic acid; lowers pH, denatures casein, gives yoghurt, cheese, sauerkraut, kimchi their characteristic acidic taste.

Final Answer: Pyruvate $\rightarrow$ lactate by LDH $\Rightarrow$ C

Answer: (C) Go Back to Q5

Q6.

Solution

Concept — Three-level folding of small intestinal mucosa: The small intestine ($\sim$ 6 m long) needs an enormous absorptive surface, achieved by nesting three orders of folding.

Step 1 — Plicae circulares (Kerckring's folds): Visible macroscopic transverse folds (a few cm tall) of the mucosa + submucosa; permanent (do not disappear when distended, unlike gastric rugae). Most prominent in jejunum. Multiply surface area $\sim 3\times$.

Step 2 — Villi: Finger-like projections of mucosa, ~ 0.5–1 mm tall. Each villus contains a central lacteal (lymphatic capillary, absorbs dietary fats as chylomicrons) and a network of blood capillaries (absorb water-soluble nutrients). Surface area multiplied $\sim 10\times$ further.

Step 3 — Microvilli (brush border): Tiny cytoplasmic projections of each enterocyte apical surface, $\sim 1\,\mu$m long. ~ 1000 microvilli per cell. Visible as the "brush border" under light microscopy. Surface area multiplied another $\sim 20\times$.

Step 4 — Net surface area: Cylinder surface (smooth tube) of intestine = $\sim 0.4\,\text{m}^2$. With all three foldings: $\sim 200\,\text{m}^2$ — a 500-fold increase, comparable to a tennis court.

Step 5 — Brush-border enzymes and absorption: Microvilli display digestive enzymes (lactase, sucrase, maltase, dipeptidases, alkaline phosphatase) and ac-

tive transporters (SGLT-1 for glucose/galactose Na^+-coupled symport; GLUT5 for fructose facilitated diffusion; PepT1 for di/tri-peptides).

Final Answer: Plicae circulares + villi + microvilli $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept — Haemoglobin (HbA) quaternary structure: First protein whose three-dimensional structure was solved by X-ray crystallography (Max Perutz, 1959; Nobel 1962).

Step 1 — Subunit composition: Adult haemoglobin A: $\alpha_2\beta_2$ — a tetramer of two α-globin chains (141 aa, encoded on chromosome 16) and two β-globin chains (146 aa, encoded on chromosome 11). The two $\alpha\beta$ dimers pack tightly into a near-spherical tetramer ($\sim 6.4\,\text{nm}$ across).

Step 2 — Haem groups: Each chain non-covalently binds one haem (iron-protoporphyrin IX) prosthetic group, with the iron coordinated by the proximal histidine. So one Hb tetramer can bind FOUR O_2 molecules.

Step 3 — Cooperative O_2 binding: The four binding sites communicate: binding of O_2 at one site triggers a T (tense, low-affinity) $\rightarrow$ R (relaxed, high-affinity) conformational shift, increasing affinity at the remaining three sites. Results in the famous sigmoidal O_2 dissociation curve.

Step 4 — Developmental haemoglobins:

- Embryonic: $\zeta_2\epsilon_2$, etc.
- Foetal: HbF ($\alpha_2\gamma_2$) — higher O_2 affinity than HbA (binds 2,3-BPG less avidly) for placental O_2 uptake.
- Adult: HbA ($\alpha_2\beta_2$, $\sim 97\%$); HbA_2 ($\alpha_2\delta_2$, $\sim 2.5\%$); HbF (residual $< 1\%$).

Step 5 — Disorders: Mutations in β-chain (sickle cell, β-thalassaemia); α-chain (α-thalassaemia); deletion of entire clusters (HPFH).

Final Answer: Tetramer $\alpha_2\beta_2$ with 4 haem groups $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept — Frank-Starling law of the heart: Independently described by Otto Frank (1895) using frog heart and Ernest Starling (1914) using dog heart-lung preparation. A foundational principle of cardiovascular physiology.

Step 1 — The law (modern statement): "The heart pumps out whatever blood is delivered to it" — within physiological limits, stroke volume (SV) increases as end-diastolic volume (EDV) increases.

Step 2 — Mechanism (length-tension relationship):

- Greater venous return $\Rightarrow$ greater EDV $\Rightarrow$ greater myocardial fibre stretch.
- Sarcomere length approaches optimum ($\sim$ $2.2\,\mu\text{m}$), where actin-myosin overlap is maximal.
- Higher Ca^{2+} sensitivity at longer length (length-dependent activation, due to changes in troponin C affinity and lattice spacing).
- Therefore stronger contractile force $\Rightarrow$ larger SV.

Step 3 — Functional significance:

- Matches output of the right and left ventricles automatically (otherwise blood would pool in pulmonary or systemic circulation).
- Allows immediate adaptation to changing venous return (exercise, postural changes) without neural or hormonal input.
- Underlies the "Starling curve" (SV vs preload), shifted by inotropic state.

Step 4 — Limits: At extreme overstretch (left ventricular failure, dilated cardiomyopathy), sarcomeres overshoot optimum length, the curve flattens or descends $\Rightarrow$ pulmonary congestion (left failure) or peripheral oedema (right failure).

Final Answer: Heart pumps out whatever venous return delivers; greater stretch $\rightarrow$ stronger contraction $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q8

Q9.

Solution

Concept — The counter-current multiplier and exchanger of the kidney: The U-shaped loop of Henle establishes a steep medullary osmotic gradient ($\sim 300\,\mathrm{mOsm}$ at cortex, ~ 1200 in deep medulla in humans; up to $3000+$ in desert rodents); ADH then exploits this gradient to concentrate urine.

Step 1 — Anatomy:

- Descending limb (thin): highly water-permeable (via aquaporin 1); impermeable to ions.
- Ascending limb (thick): water-impermeable; actively reabsorbs Na^+, K^+, Cl^- via the Na/K/2Cl symporter (NKCC2) into the interstitium.
- Vasa recta: capillary loops running parallel to the loop of Henle.

Step 2 — Counter-current multiplier (the loop itself): The active salt pumping from the ascending limb deposits NaCl in the medullary interstitium, raising its osmolarity. Water is then drawn osmotically out of the descending limb (water-permeable), concentrating the tubular fluid. The longer the loop, the more the salt is "multiplied" along the gradient. Net effect: the medullary interstitium becomes very salty.

Step 3 — Counter-current exchanger (vasa recta): Without a special arrangement, blood flowing through the medulla would wash out the gradient. The vasa recta solves this by running parallel U-shaped loops:

- Descending vasa recta: blood picks up salt (and loses water) as it descends.
- Ascending vasa recta: blood loses salt (and picks up water) as it ascends.
- Net: salt is preserved in the medulla; only a small fraction is washed out.

Step 4 — ADH-mediated urine concentration:

- When dehydrated, ADH (vasopressin) from posterior pituitary inserts aquaporin-2 channels into collecting duct apical membrane.
- Collecting duct now becomes water-permeable; water moves out into the salty medullary interstitium, leaving concentrated urine.
- Without ADH (water excess), collecting duct stays impermeable, dilute urine is produced.

Step 5 — Urea recycling: Urea contributes $\sim 50\%$ of medullary osmolarity; recycled between collecting duct and loop of Henle via UT1/UT2 urea transporters.

Step 6 — Desert mammal adaptation: Kangaroo rats have very long loops of Henle, allowing them to concentrate urine to $\sim 6000\,\mathrm{mOsm}$ — they can survive without drinking water, getting all needed water from food.

Final Answer: Counter-current multiplier (loop) + exchanger (vasa recta) $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept — The neuromuscular junction: A specialised chemical synapse between the axon terminal of a somatic motor neuron and a skeletal muscle fibre. Site of much clinical relevance (myasthenia gravis, curare, succinylcholine, organophosphate poisoning).

Step 1 — Sequence of events:

(a) Action potential reaches motor nerve terminal.
(b) Voltage-gated Ca^{2+} channels open; Ca^{2+} influx triggers exocytosis of synaptic vesicles loaded with acetylcholine (ACh).
(c) ACh crosses the $\sim 50\,\mathrm{nm}$ synaptic cleft and binds nicotinic ACh receptors (nAChR) on the motor end-plate of the muscle membrane.
(d) nAChR is a pentameric ($\alpha_2\beta\gamma\delta$) ligand-gated cation channel; binding of two ACh molecules opens the central pore.
(e) Na^+ enters (some K^+ exits), depolarising the end-plate $\rightarrow$ end-plate potential.
(f) EPP propagates and triggers a muscle action potential, ultimately Ca^{2+} release from sarcoplasmic reticulum $\rightarrow$ contraction.

Step 2 — ACh removal: Acetylcholinesterase (AChE) in the cleft rapidly hydrolyses ACh $\rightarrow$ acetate + choline; choline is taken back up into the nerve terminal for re-synthesis of ACh. This rapid clearance ensures discrete signalling.

Step 3 — Pharmacology of the NMJ:

- Non-depolarising blockers: curare, vecuronium, atracurium — competitive antagonists of nAChR; used in surgical muscle relaxation.
- Depolarising blockers: succinylcholine — agonist that causes prolonged depolarisation $\rightarrow$ flaccid paralysis.
- AChE inhibitors: neostigmine, pyridostigmine (treat myasthenia gravis); donepezil (Alzheimer's, on central cholinergic system); organophosphate insecticides and nerve agents (sarin, VX) — toxic.

- α-Bungarotoxin (cobra venom): irreversibly binds nAChR.
- Botulinum toxin (Botox): blocks ACh release from nerve terminal.
- Myasthenia gravis: autoantibodies against nAChR $\rightarrow$ muscle weakness, fatigability.

Step 4 — Distinguish from autonomic ganglion (also nicotinic) vs. parasympathetic effector (muscarinic) ACh receptors: Nicotinic AChR: ligand-gated ion channel; fast; at NMJ + autonomic ganglia. Muscarinic AChR: GPCR; slower; at parasympathetic effector organs.

Final Answer: Nicotinic acetylcholine receptors $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept — Parathyroid hormone (PTH) — the master calcium regulator: An 84-amino-acid peptide secreted by chief cells of the four parathyroid glands (embedded in posterior thyroid). The principal hormone that defends against hypocalcaemia.

Step 1 — Stimulus and sensing: Plasma Ca^{2+} is monitored by the calcium-sensing receptor (CaSR), a GPCR on the parathyroid chief cells. Low Ca^{2+} $\Rightarrow$ less CaSR activation $\Rightarrow$ PTH release (counterintuitive negative feedback).

Step 2 — Three target tissues, each raising plasma Ca^{2+}:

(a) **Bone:** PTH binds receptors on osteoblasts $\rightarrow$ they produce RANKL $\rightarrow$ activates osteoclast precursors $\rightarrow$ osteoclast-mediated bone resorption $\rightarrow$ Ca^{2+} and phosphate released into blood.

(b) **Kidney:** PTH increases Ca^{2+} reabsorption in DCT (via TRPV5 channel) and decreases phosphate reabsorption in PCT (excretes PO_4^{3-} in urine to prevent $CaPO_4$ precipitation). Also activates 1α-hydroxylase, converting 25-OH-vit D to $1,25$-$(OH)_2$-vit D (calcitriol).

(c) **Gut (indirectly via calcitriol):** calcitriol upregulates intestinal Ca^{2+} uptake (TRPV6) and binding proteins (calbindin).

Step 3 — Counter-regulatory hormone — calcitonin: Secreted by parafollicular C cells of thyroid in response to high Ca^{2+}; inhibits osteoclasts; lowers blood Ca^{2+}. Of relatively minor importance in humans (thyroidectomised patients have normal Ca^{2+}).

Step 4 — Disorders:

- Hyperparathyroidism: hypercalcaemia, kidney stones, bone resorption ("stones, bones, abdominal groans, psychic moans").
- Hypoparathyroidism (often surgical, post-thyroidectomy): hypocalcaemia, tetany (Trousseau's, Chvostek's signs), seizures.
- Renal failure: low $1{,}25$-$(OH)_2$-vit D $\rightarrow$ secondary hyperparathyroidism $\rightarrow$ renal osteodystrophy.
- Rickets / osteomalacia: vitamin D deficiency.

Final Answer: Bone resorption + renal Ca^{2+} reabsorption + calcitriol activation $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q11

Q12.

Solution

Concept — Cyclic vs non-cyclic (linear) electron flow: The light reactions of photosynthesis can route electrons in two modes; chloroplasts switch between them according to metabolic need.

Step 1 — Non-cyclic (linear) electron flow (the Z-scheme):

- PS II splits water: $2\,H_2O \rightarrow 4\,H^+ + 4\,e^- + O_2$; electrons enter ETC.
- Plastoquinone $\rightarrow$ cytochrome b_6f complex (pumps H^+ into thylakoid lumen) $\rightarrow$ plastocyanin $\rightarrow$ PS I.
- PS I excited again by light $\rightarrow$ ferredoxin $\rightarrow$ $NADP^+$ reductase $\rightarrow$ **NADPH**.
- Proton gradient drives ATP synthase $\rightarrow$ ATP.
- Products: NADPH + ATP + O_2.

Step 2 — Cyclic electron flow (cyclic photophosphorylation):

- Only PS I operates; PS II inactive.
- Electrons cycle from PS I $\rightarrow$ ferredoxin $\rightarrow$ cytochrome b_6f $\rightarrow$ plastocyanin $\rightarrow$ back to PS I.
- This still pumps H^+ into the lumen (via b_6f), so still drives ATP synthesis.
- But NO water splitting, NO O_2 evolution, NO NADPH.
- Net product: ATP only.

Step 3 — Why have cyclic flow? The Calvin cycle consumes ATP and NADPH in a $3:2$ ratio (typically 3 ATP and 2 NADPH per CO_2 fixed). Linear flow produces them in roughly $1:1$. The extra ATP demand is met by cyclic flow. Especially important in C_4 plants where extra ATP is needed for the C_4 carbon-concentrating pump.

Step 4 — Discovery: Daniel Arnon (1954) demonstrated cyclic photophosphorylation in isolated spinach chloroplasts, distinguishing it from oxygen-evolving (non-cyclic) photophosphorylation.

Final Answer: Only PS I; electrons cycle back to PS I; only ATP produced $\Rightarrow$ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept — Chi-square goodness-of-fit test: A statistical test devised by Karl Pearson (1900); the standard for testing whether observed categorical data deviate significantly from expectation.

Step 1 — The formula:

$$\chi^2 = \sum_{i=1}^{k} \frac{(O_i - E_i)^2}{E_i}$$

where O_i is the observed count in class i, E_i is the expected count, and the sum runs over all k classes.

Step 2 — Example — testing $3:1$ Mendelian ratio: Cross of Aa $\times$ Aa gives expected $3:1$ (dominant : recessive). Suppose we score 400 offspring: 290 dominant, 110 recessive.

- Expected: $E_{dom} = 400 \times 3/4 = 300$; $E_{rec} = 400 \times 1/4 = 100$.
- $\chi^2 = (290-300)^2/300 + (110-100)^2/100 = 100/300 + 100/100 = 0.33 + 1.00 = 1.33$.
- Degrees of freedom = (number of classes) $-1 = 2 - 1 = 1$.
- Critical $\chi^2_{0.05,df=1} = 3.84$.
- Calculated χ^2 (1.33) $<$ critical (3.84) $\Rightarrow$ fail to reject null hypothesis $\Rightarrow$ data are consistent with $3:1$.

Step 3 — p-value interpretation: $p > 0.05$: deviation from expected is consistent with sampling chance. $p < 0.05$: reject null hypothesis; observed pattern unlikely under expected ratio.

Step 4 — Caveats: χ^2 requires expected counts ≥ 5 in each class (otherwise use Fisher's exact test); not suitable for very small samples or for testing means/variances (use t-test, ANOVA).

Final Answer: Chi-square (χ^2) goodness-of-fit test $\Rightarrow$ A

Answer: (A) Go Back to Q13

Q14.

Solution

Concept — Polygenic (quantitative) inheritance: Mendel's laws were derived from discrete (qualitative) traits with few alleles per locus. Many real-world traits show continuous variation — they are governed by many genes with additive small effects.

Step 1 — Nilsson-Ehle's wheat-kernel-colour experiment (1909): Crossed dark-red and white wheat. F_1 all intermediate red. F_2 showed not a sharp ratio but a continuous distribution from white (1/64) through five shades of red to dark red (1/64). Interpretation: **three independent gene loci**, each with two alleles (capital R contributing some red pigment, lowercase r contributing none); phenotype determined by the number of capital R alleles across all loci (0 to 6).

Step 2 — Features of polygenic inheritance:

- Several gene loci involved (often 5–20).
- Each locus has alleles that contribute additively to the trait.
- Phenotypic distribution approaches a normal (Gaussian) bell curve.
- Environmental factors usually modulate further (nutrition, climate).
- No sharp dominance; intermediate phenotypes blur classes.

Step 3 — Human examples:

- **Skin colour:** $\sim$ 4–8 genes (MC1R, OCA2, TYRP1, SLC24A5, etc.); melanin amount determined by total "dose".
- **Height:** $\geq$ 700 loci identified by GWAS, each contributing a few mm; heritability $\sim 80\%$.
- **Intelligence (IQ):** highly polygenic and environment-influenced.
- **Blood pressure, BMI, diabetes risk:** polygenic.

Step 4 — Quantitative trait loci (QTL): The genomic regions whose variation affects a quantitative trait. Mapped statistically by linkage analysis or GWAS. Personal genomic risk scores (PRS) sum many small-effect SNPs to predict polygenic disease risk.

Final Answer: Additive effects of many genes plus environment $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q14

Q15.

Solution

Concept — Post-translational modifications (PTMs): After ribosomal synthesis, most proteins are covalently modified, expanding the $\sim 20{,}000$ human genes into a proteome of > 1 million distinct protein species.

Step 1 — Major reversible PTMs:

- **Phosphorylation:** P added to S, T, Y residues by kinases; removed by phosphatases. Toggles activity in receptor tyrosine kinase signalling (insulin receptor, EGFR), MAP kinase cascades, cell-cycle CDKs.
- **Acetylation:** on lysines; histone acetylation (by HATs) loosens chromatin and activates transcription; HDACs remove it.
- **Methylation:** on lysines, arginines; histone methylation can activate or repress depending on residue.
- **Ubiquitination:** adds ubiquitin chains; tags proteins for $26S$ proteasomal degradation; also signals (mono-ubiquitination affects trafficking, repair).
- **Glycosylation:** sugars added (covered in Q3).
- **Hydroxylation:** of prolines and lysines in collagen by prolyl/lysyl hydroxylases (vit C-dependent).
- **Lipidation:** prenylation (Ras), myristoylation, palmitoylation — attaches proteins to membranes.

Step 2 — The "histone code": Histone tails carry dozens of distinct modifications. Combinations (H3K4me3, H3K27ac, H3K9me3, etc.) form a chromatin-state code read by "reader" protein domains (bromodomains for ac, chromodomains for methyl), recruiting transcription factors and chromatin remodellers.

Step 3 — Why PTMs matter:

- Fast (seconds-minutes) regulation, much faster than transcriptional response.
- Reversible; one protein can be toggled on/off many times.
- Combinatorial diversity (one protein may have $10+$ phosphosites, each independently regulated).
- Drug targets: kinase inhibitors (imatinib for BCR-ABL; gefitinib for EGFR), histone deacetylase inhibitors (vorinostat), proteasome inhibitors (bortezomib for myeloma).

Final Answer: Reversibly modify activity, stability, localisation, interactions, degradation $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q15

Q16.

Solution

Concept — The *trp* operon — a repressible (anabolic) operon: A cluster of 5 genes encoding the enzymes for tryptophan biosynthesis (*trpE, D, C, B, A*). Discovered by Charles Yanofsky and contributes alongside Jacob-Monod *lac* model to our understanding of gene regulation.

Step 1 — Why repressible? Tryptophan is an end-product the bacterium synthesises if needed; if Trp is plentiful from the environment, making it anew would be wasteful. So the operon should be ON when Trp is scarce, and OFF when Trp is abundant — the inverse logic of *lac* (which is induced by lactose).

Step 2 — Mechanism — the Trp repressor:

- Encoded by *trpR* (a separate regulatory gene).
- By itself, Trp repressor is INACTIVE — it cannot bind the operator.
- When intracellular tryptophan is high, Trp binds the repressor (as **co-repressor**) $\rightarrow$ allosteric activation $\rightarrow$ Trp-repressor complex binds the operator (overlapping the promoter) $\rightarrow$ blocks RNA polymerase $\rightarrow$ transcription OFF.
- When Trp is low, repressor is inactive; operon is ON; enzymes are made; Trp is synthesised.

Step 3 — A second layer: attenuation: The *trp* operon also has a more refined fine-tuning mechanism called **attenuation**, mediated by a leader sequence containing several tryptophan codons. Translation of the leader peptide stalls (when Trp-tRNA is scarce) or proceeds (when Trp-tRNA is abundant), causing the nascent mRNA to fold into either an antiterminator or a terminator hairpin — modulating whether transcription continues into the structural genes. This adds a translation-coupled sensor of intracellular tryptophan supply.

Step 4 — Compare repressible vs inducible operons:

	trp (repressible)	*lac* (inducible)
Pathway type	anabolic	catabolic
Default state	ON	OFF
Effector	Trp (co-repressor)	lactose/allolactose (inducer)
Effector role	activates repressor	inactivates repressor

Final Answer: Repressible operon; Trp acts as co-repressor $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q16

Q17.

Solution

Concept — Origin of life: the Oparin-Haldane hypothesis tested: Alexander Oparin (1924) and J.B.S. Haldane (1929) independently proposed that life on early Earth arose by chemical evolution from inorganic precursors in a reducing atmosphere (rich in H_2, CH_4, NH_3, H_2O) with energy from lightning and UV.

Step 1 — The classic Miller-Urey experiment (1952): Stanley Miller (graduate student) and Harold Urey (his advisor, 1934 Nobel laureate) at University of Chicago set up:

- A sealed flask with the four "primitive atmosphere" gases: CH_4, NH_3, H_2, H_2O.
- Electric sparks between tungsten electrodes (simulating lightning).
- A condenser to liquefy water vapour and return liquid to the base of the apparatus (simulating rain into the "primitive ocean").
- Heated the water to maintain circulation.
- Ran for one week.

Step 2 — Results: The water became reddish-brown. Chemical analysis revealed:

- Several proteinogenic amino acids: glycine, alanine, α-aminobutyric acid, aspartic acid.
- Other organic molecules: hydroxy acids, urea, formic acid, simple sugars.
- Re-analysis decades later (with better techniques) found over 20 amino acids and even nucleobase precursors.

Step 3 — Significance: Established that the building blocks of life (amino acids, simple organics) can be produced abiotically from simple gases under conditions plausible for early Earth. **Did NOT create life** — only the monomers.

Step 4 — Modern updates:

- Re-analysis of original Miller samples (2008) showed even more amino acids than originally reported.
- Some scientists believe the early Earth's atmosphere may have been more neutral (CO_2, N_2, H_2O) rather than strongly reducing, requiring catalysts or alternative settings (hydrothermal vents).
- Murchison meteorite contains ~ 70 amino acids of extraterrestrial origin — showing prebiotic chemistry happens widely in the cosmos.
- Polymerisation of monomers, formation of cells (protocells), and RNA-world emergence remain active research areas.

Final Answer: Amino acids form spontaneously from reducing atmosphere + spark energy $\Rightarrow$ B

Answer: (B) Go Back to Q17

Q18.

Solution

Concept — Zona pellucida and sperm-egg recognition: The zona pellucida (ZP) is a glycoprotein-rich extracellular matrix surrounding the mammalian oocyte. Acts as the "species-specific gatekeeper" for sperm.

Step 1 — ZP composition (mouse/human): 3–4 glycoproteins (ZP1, ZP2, ZP3, sometimes ZP4) secreted by the growing oocyte itself (and possibly granulosa cells). ZP2 and ZP3 form long filaments crosslinked by ZP1.

Step 2 — ZP3 — the primary sperm receptor: Sperm acrosomal proteins (e.g. zonadhesin, sp56, β-1,4-galactosyltransferase) recognise specific O-linked carbohydrates on ZP3. This species-specific binding ensures that human sperm fertilise only human eggs, mouse only mouse, etc.

Step 3 — Triggering the acrosome reaction: Once bound to ZP3:

- ZP3 binding activates a signal cascade (Ca^{2+} entry, G-protein involvement) in the sperm head.
- The acrosomal membrane fuses with the sperm plasma membrane, releasing hydrolytic enzymes: hyaluronidase (cleaves cumulus matrix), acrosin (a serine protease that cleaves ZP2).
- The sperm bores its way through the zona pellucida toward the egg's plasma membrane.

Step 4 — ZP2 — the secondary receptor: After the acrosome reaction, sperm proteins engage ZP2, holding the sperm tightly to the zona while it tunnels through.

Step 5 — The cortical reaction and the block to polyspermy: Once a single sperm fuses with the oocyte plasma membrane:

- Ca^{2+} waves spread through the egg cytoplasm.
- Cortical granules just beneath the oocyte plasma membrane fuse and release proteases.
- These cleave ZP2 $\rightarrow$ hardens the zona, makes it impermeable to further sperm.

- ZP3 is also altered to lose sperm-binding ability.
- Result: only one sperm fertilises the egg (the block to polyspermy).

Final Answer: ZP3 glycoprotein in zona pellucida $\Rightarrow$ C

Answer: (C) Go Back to Q18

Q19.

Solution

Concept — Early human embryonic development: A precise sequence of cleavages and differentiations transforms the single-celled zygote into the implanting blastocyst in 5–7 days.

Step 1 — Day-by-day:

- Day 0: fertilisation in fallopian tube ampulla $\rightarrow$ zygote (1 cell).
- Day 1: first cleavage $\rightarrow$ 2 cells.
- Day 2: 4 cells.
- Day 3: 8 cells; compaction begins (cells maximise surface contact via E-cadherin).
- Day 4: morula ($\sim$ 16-cell solid ball; Latin *morus* = mulberry); still within zona pellucida.
- Day 5: cavitation $\rightarrow$ blastocoel forms; cells differentiate into inner cell mass (ICM) and trophoblast layer $\rightarrow$ **blastocyst**.
- Day 6–7: blastocyst hatches from the zona pellucida.
- Day 7–9: blastocyst implants into the endometrial wall of the uterus.

Step 2 — Blastocyst anatomy:

- **Trophoblast (trophectoderm):** outer layer, $\sim$ 100 cells; will become the chorion, placenta, foetal membranes.
- **Inner cell mass (ICM, embryoblast):** $\sim$ 30 cells clumped at one pole; will give rise to the embryo proper and yolk sac.
- **Blastocoel:** fluid-filled cavity.

Step 3 — Why this matters:

- Implantation occurs at the blastocyst stage, not earlier.
- Embryonic stem cells (ESCs) are derived from the ICM of human blastocysts ($\sim$ 100-cell stage). They are pluripotent: can give rise to any of the three germ layers.

- IVF embryos are typically transferred at blastocyst stage (day 5) for higher implantation rates than at day-3 cleavage stage.
- Pre-implantation genetic testing (PGT) typically biopsies ~ 5 cells from the trophoblast on day 5.

Step 4 — Distinguish from gastrula: Gastrulation begins in week 3 post-implantation, when the embryonic disk forms the three primary germ layers (ectoderm, mesoderm, endoderm) by ingression through the primitive streak.

Final Answer: Blastocyst $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q19

Q20.

Solution

Concept — Male reproductive endocrine axis: The hypothalamic-pituitary-testicular axis is regulated by two anterior pituitary gonadotropins: FSH and LH.

Step 1 — FSH actions in males:

- Acts ONLY on Sertoli cells (which carry the FSH receptor); Leydig cells do NOT respond to FSH.
- Stimulates spermatogenesis (initiation in puberty; maintenance in adulthood).
- Stimulates Sertoli cell production of androgen-binding protein (ABP), which concentrates testosterone in the seminiferous tubules (essential for spermatogenesis).
- Stimulates inhibin secretion, which feeds back negatively on the anterior pituitary to inhibit further FSH release.

Step 2 — LH actions in males:

- Acts on Leydig (interstitial) cells.
- Stimulates cholesterol uptake, steroidogenic enzyme expression, and testosterone synthesis.
- Testosterone has both local (intratubular) and systemic effects.

Step 3 — Feedback regulation:

- Hypothalamic GnRH (pulsatile) drives anterior pituitary FSH and LH secretion.

- Testosterone (from Leydig) feeds back negatively on hypothalamus (GnRH) and pituitary (LH).
- Inhibin (from Sertoli) feeds back negatively on pituitary (FSH specifically).

Step 4 — Clinical relevance:

- Hypogonadotropic hypogonadism: low FSH/LH $\rightarrow$ low testosterone + low sperm $\rightarrow$ infertility.
- Klinefelter (47,XXY): seminiferous tubule failure; high FSH/LH (compensatory) but low testosterone.
- Anabolic steroid abuse: exogenous testosterone suppresses hypothalamus-pituitary $\rightarrow$ low LH and FSH $\rightarrow$ low intratubular testosterone $\rightarrow$ azoospermia and testicular atrophy.

Step 5 — Comparison with female: In females, FSH stimulates granulosa cells of the ovarian follicle (analogous to Sertoli cells). LH stimulates theca cells (analogous to Leydig). Same molecular hormones, different target cells in the gonad.

Final Answer: Sertoli cells of seminiferous tubules $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q20

Q21.

Solution

Concept — Combined oral contraceptive pill (COCP): Developed by Gregory Pincus and colleagues; first approved 1960 as Enovid. Among the most studied medications in history. $\sim$ 100 million women use it daily worldwide.

Step 1 — Composition:

- Synthetic oestrogen (typically ethinyl oestradiol).
- Synthetic progestin (levonorgestrel, desogestrel, drospirenone, etc.).
- Taken daily for 21 days, with 7-day pill-free interval (withdrawal bleed).

Step 2 — Triple mechanism of action:

(a) **Inhibits ovulation (primary):** continuous oestrogen + progestin exerts strong negative feedback on hypothalamus (GnRH) and anterior pituitary, suppressing FSH and especially the mid-cycle LH surge $\rightarrow$ no ovulation.
(b) **Thickens cervical mucus:** progestin makes mucus viscous, hostile to sperm.
(c) **Endometrial atrophy:** progestin prevents endometrium becoming receptive to implantation (even if a fertilised egg arrived).

Step 3 — Efficacy:

- Perfect use: $> 99\%$.
- Typical use: $\sim 91\%$ (missed pills, drug interactions).

Step 4 — Non-contraceptive benefits:

- Reduced menstrual blood loss, dysmenorrhoea.
- Regularised cycles (helpful for PCOS).
- Decreased risk of ovarian, endometrial, and colorectal cancers.
- Treatment of acne, hirsutism.

Step 5 — Risks: Small increased risk of venous thromboembolism (especially with smoking, age > 35), slight cardiovascular risk increase, small increased breast cancer risk. Contraindications: history of thrombosis, migraine with aura, uncontrolled hypertension.

Final Answer: Suppresses ovulation by negative feedback on HPG axis $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q21

Q22.

Solution

Concept — Parthenocarpy: Greek *parthenos* = virgin + *karpos* = fruit. The development of fruit without fertilisation; consequently the fruit is seedless (or seeds are abortive).

Step 1 — Types of parthenocarpy:

- **Vegetative (obligate):** fruit develops without any pollination at all (banana, pineapple, navel orange).
- **Stimulative parthenocarpy:** pollination is required to trigger fruit development, but fertilisation does not occur (some figs, some seedless grapes).
- **Stenospermocarpy:** fertilisation occurs but seeds abort early (Thompson seedless grapes, watermelon 'seedless' varieties).

Step 2 — Mechanism: Normally, fertilisation triggers hormone release (auxin, gibberellins) from the developing seed, signalling the ovary to expand into a fruit. In parthenocarpy:

- Either the ovary tissue spontaneously produces these hormones independent of fertilisation,

- Or external hormone application can trigger fruit development.

Step 3 — Commercial induction:

- Spray auxin (NAA, 2,4-D) or gibberellin (GA_3) onto unpollinated flowers $\rightarrow$ induces parthenocarpic fruit set in many species (tomato, brinjal, watermelon).
- Especially useful when conditions are unfavourable for pollination (greenhouse cultivation, cold weather, lack of insect pollinators).

Step 4 — Examples and commercial importance:

- Banana (triploid sterile cultivars): seedless via parthenocarpy from infertile $3n$ genotypes.
- Pineapple: stimulative parthenocarpy.
- Citrus: navel orange, satsuma mandarin.
- Grapes: many seedless varieties.
- Watermelon: induced triploids give seedless fruit.
- Tomato: parthenocarpic varieties grown in greenhouses where pollinators are scarce.

Final Answer: Fruit development without fertilisation $\rightarrow$ seedless fruit $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q22

Q23.

Solution

Concept — Cytokinins: Discovered by Folke Skoog and Carlos Miller (1955) when they identified "kinetin" from autoclaved herring sperm DNA — a degradation product (N6-furfuryladenine) that promoted cell division in tobacco pith cultures. Natural cytokinins (zeatin, isopentenyladenine) are adenine derivatives.

Step 1 — Major effects:

- **Cell division:** stimulates cytokinesis (the original "cyto-kinin" name). Required (with auxin) for cell-cycle progression in plant tissue cultures.
- **Shoot regeneration in tissue culture:** the Skoog-Miller experiments showed that the ratio of cytokinin to auxin in the medium controls morphogenesis:
  - High cytokinin / low auxin $\rightarrow$ shoot formation.
  - Low cytokinin / high auxin $\rightarrow$ root formation.

  - Roughly equal → undifferentiated callus growth.
- **Counteracts apical dominance:** promotes axillary bud outgrowth (the opposite of auxin's effect from the apical bud).
- **Delays senescence:** keeps leaves green (→ the "Richmond-Lang effect"; cytokinin treatment can keep detached leaves green for weeks).
- **Promotes nutrient mobilisation:** cytokinin-rich tissue acts as a sink, drawing nutrients.
- **Chloroplast development:** promotes chloroplast biogenesis and chlorophyll synthesis.

Step 2 — Site of synthesis: Primarily in root tips; transported in xylem to shoot. Local synthesis also in young leaves and developing fruits.

Step 3 — Practical applications:

- Tissue culture / micropropagation: cytokinin-rich media induce multiple shoots from a single explant.
- Cut flower preservation: floral preservatives often contain a cytokinin to delay senescence.
- Crop yield: foliar cytokinin sprays can delay leaf senescence, prolonging the photosynthetic period.

Final Answer: Promote cytokinesis, lateral bud outgrowth, delay senescence, shoot regeneration ⇒ A

Answer: (A) Go Back to Q23

Q24.

Solution

Concept — Gravitropism (geotropism): The directional growth of a plant in response to gravity. Roots grow downward (positively gravitropic); primary shoots grow upward (negatively gravitropic).

Step 1 — Gravity perception — the statolith hypothesis: Specialised cells contain dense, starch-filled amyloplasts called **statoliths**.

- In roots: located in columella cells of the root cap.
- In shoots: located in endodermis cells (the starch sheath).

When the plant is reoriented, the statoliths sediment under gravity to the new bottom of the cell, contacting the lower wall and triggering signalling.

Step 2 — The Cholodny-Went hypothesis (gravity-induced auxin asymmetry):

- Statolith sedimentation causes the redistribution of PIN auxin-efflux carriers to the lower side of the cell.
- Auxin (IAA) accumulates on the lower side.
- In SHOOTS: high auxin = more elongation $\rightarrow$ lower side grows faster $\rightarrow$ shoot bends upward. NEGATIVE gravitropism.
- In ROOTS: high auxin = LESS elongation (roots are very sensitive; even low auxin inhibits) $\rightarrow$ lower side grows slower $\rightarrow$ root bends downward. POSITIVE gravitropism.

Step 3 — Signal transduction:

- pH changes (acidic on growing side).
- Ca^{2+} fluxes.
- PIN protein cycling between plasma membrane and endosomes.

Step 4 — Other tropisms (for comparison):

- Phototropism (response to light): shoot grows toward light (auxin redistribution, mediated by phototropin).
- Thigmotropism (response to touch): tendrils coil around supports.
- Hydrotropism (response to water): roots grow toward moisture.
- Chemotropism (response to chemicals): pollen tubes grow toward ovule chemoattractants.

Step 5 — Spaceflight experiments: On the ISS, plants show altered growth and orientation (reduced statolith-based gravity sensing) but can still grow, using phototropism and other cues.

Final Answer: Gravitropism (statolith-based, auxin-mediated) $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q24

Q25.

Solution

Concept — Heterocysts and the nitrogen-oxygen problem: Some filamentous cyanobacteria (*Anabaena*, *Nostoc*, *Calothrix*) fix atmospheric N_2 into ammonia using the enzyme nitrogenase. The challenge: nitrogenase is irreversibly destroyed by oxygen, but the same cells produce O_2 during photosynthesis. The solution: **compartmentalisation in heterocysts**.

Step 1 — Heterocyst features:

- Larger than vegetative cells; thicker, multi-layered envelope (glycolipid outer wall, polysaccharide inner wall) restricting O_2 entry.
- Lacks Photosystem II (which generates O_2 from water splitting); retains only Photosystem I (cyclic photophosphorylation $\rightarrow$ ATP without O_2).
- Elevated respiration consumes any residual O_2.
- Contains active nitrogenase, which fixes atmospheric N_2 to ammonia using $8\,H^+$, $8\,e^-$, and 16 ATP per N_2, yielding $2\,NH_3 + H_2$.
- Receives carbohydrates from neighbouring photosynthetic vegetative cells (through cell-cell connections), and supplies them with fixed nitrogen (glutamine).

Step 2 — Spatial distribution: Regularly spaced along the filament (every 10–20 vegetative cells), forming a quasi-periodic pattern. The patterning is controlled by a signalling system: HetN/PatS inhibitors diffusing from differentiating heterocysts prevent neighbours from also differentiating.

Step 3 — Ecological importance:

- Major biological nitrogen fixers in freshwater, marine, and soil ecosystems.
- *Anabaena azollae* lives symbiotically in cavities of the leaves of *Azolla* water fern — used as a green manure in rice paddies to boost yield.
- Some lichens contain cyanobionts (*Nostoc*) that contribute fixed nitrogen.

Step 4 — Akinetes and hormogonia: Other specialised cells in cyanobacteria: akinetes (thick-walled resting spores), hormogonia (short motile filaments for dispersal).

Final Answer: Heterocysts (O_2-protected, nitrogen-fixing specialised cells) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q25

Q26.

Solution

Concept — Lichen — a composite organism: Beatrix Potter (the children's author!) and Simon Schwendener proposed in the 1860s that lichens are dual organisms — now established as mutualistic symbioses.

Step 1 — Components:

- **Mycobiont** (fungal partner): usually an Ascomycete (rarely Basidiomycete); forms most of the lichen thallus (the body); provides structure, holdfast, water retention, mineral uptake, UV protection. Cannot live alone in nature.
- **Photobiont** (photosynthetic partner): either a green alga (e.g. *Trebouxia*, the most common), a cyanobacterium (e.g. *Nostoc* — in some lichens this provides both photosynthesis AND nitrogen fixation), or sometimes both in the same lichen. Provides photosynthate (sugars, polyols) to the fungus.

Step 2 — Three growth forms:

- **Crustose:** crust-like, flat, intimately attached to substrate (rocks, tree bark). Slow growing.
- **Foliose:** leaf-like, with distinct upper and lower surfaces, partially attached by rhizines.
- **Fruticose:** bushy, branching, or hanging (e.g. "old man's beard" *Usnea*, reindeer moss *Cladonia*).

Step 3 — Ecological roles:

- **Pioneer organisms:** colonise bare rock in primary succession, slowly weathering rock to form soil over centuries.
- **Food** for reindeer, caribou (lichens are the main winter food in tundra).
- **Bioindicators of air pollution:** lichens are exquisitely sensitive to sulphur dioxide and acid rain $\Rightarrow$ presence/absence indicates air quality. In industrial cities, lichens often disappear.
- Nitrogen fixation: cyanolichens contribute to ecosystem N budgets.
- Antibiotic and dye production (orcein, litmus pigment from *Roccella*).

Step 4 — Reproduction:

- Vegetative (most common): isidia, soredia (small fragments containing both partners).
- Sexual: the fungal partner forms ascocarps; the resulting spores must find a compatible photobiont to establish a new lichen.

Final Answer: Symbiotic association of fungus and alga/cyanobacterium $\Rightarrow$ A

Answer: (A) Go Back to Q26

Q27.

Solution

Concept — Phylum Mollusca: Second-largest animal phylum after Arthropoda. Greek *molluscus* = soft. $\sim 85{,}000$ described species; vast diversity from microscopic snails to giant squid (largest invertebrate).

Step 1 — Body plan:

- Soft, unsegmented body (in most classes).
- Three regions: head (with sensory organs), muscular foot (locomotion), visceral mass (organs).
- Covered by a **mantle** — a fold of dorsal body wall that secretes the shell (when present) and encloses the mantle cavity housing gills (or lungs in pulmonates).
- Open circulatory system (except cephalopods — closed).

Step 2 — Diagnostic features:

- **Radula:** a chitinous ribbon-like rasping organ with rows of microscopic teeth, used to scrape food. Unique to molluscs (except bivalves, which are filter feeders).
- **Calcareous shell:** secreted by the mantle epithelium, made of calcium carbonate in protein matrix. Single (gastropods), bivalved (bivalves), internal/reduced (cephalopods), or absent (slugs, octopus).
- Gills (ctenidia) in mantle cavity for aquatic respiration; lung-like vascularised mantle for terrestrial pulmonate snails.
- True coelom (reduced to pericardial and gonadal cavities).

Step 3 — Major classes:

- **Gastropoda** (snails, slugs, limpets, abalones): largest class; torsion (twisting) during development; spiral shell.
- **Bivalvia** (Pelecypoda): clams, mussels, oysters, scallops; hinged two-part shell; filter feeders via gills; no radula.
- **Cephalopoda:** octopus, squid, cuttlefish, nautilus; highly developed nervous system, camera eyes, closed circulation, jet propulsion, sophisticated behaviour. Largest invertebrates (colossal squid, $14\,\mathrm{m}$ long).

- **Polyplacophora** (chitons): eight-plate dorsal shell.
- **Scaphopoda** (tusk shells), **Monoplacophora**, **Aplacophora**: minor groups.

Step 4 — Economic and biological importance:

- Food (oysters, mussels, clams, scallops, escargots, calamari).
- Pearls (from oysters: nacre around an irritant).
- Mother-of-pearl, sea silk, dye.
- Schistosomiasis vectors (freshwater snails).
- Octopuses and squid serve as model organisms for neuroscience.

Final Answer: Soft unsegmented body, mantle, shell, radula $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q27

Q28.

Solution

Concept — Dengue fever: A rapidly expanding tropical/subtropical arbovirus disease. Endemic in > 100 countries; estimated $\sim$ 100–400 million infections annually.

Step 1 — Pathogen — DENV:

- Family Flaviviridae, genus *Flavivirus* (close relatives: yellow fever, Zika, West Nile, Japanese encephalitis).
- Single-stranded, positive-sense RNA genome (~ 11 kb), enveloped.
- Four antigenically distinct serotypes: DENV-1, DENV-2, DENV-3, DENV-4. Infection with one gives lifelong immunity to that serotype but only short-lived cross-protection against others.

Step 2 — Vectors:

- Primary: female ***Aedes aegypti*** mosquito — highly anthropophilic, day-biting, breeds in clean stagnant water in artificial containers (tyres, flower pots, water tanks).
- Secondary: *Aedes albopictus* (Asian tiger mosquito) — expanding range, more cold-tolerant.

Step 3 — Clinical spectrum:

- **Classic dengue fever:** "break-bone fever" — abrupt onset, high fever ($40\,^\circ$C), severe headache (especially retro-orbital), myalgia, arthralgia, maculo-papular rash, thrombocytopenia, leukopenia.

- **Dengue haemorrhagic fever (DHF):** more severe; plasma leakage (pleural effusion, ascites), thrombocytopenia, haemorrhagic manifestations.
- **Dengue shock syndrome (DSS):** circulatory collapse from severe plasma leakage; potentially fatal.

Step 4 — Antibody-dependent enhancement (ADE): Sub-neutralising antibodies from a previous infection (with a different DENV serotype) can paradoxically enhance entry of new DENV into Fc-receptor-bearing cells $\rightarrow$ more severe disease in secondary infections. This is a major obstacle to vaccine development.

Step 5 — Diagnosis and management:

- Diagnosis: NS1 antigen (early), IgM/IgG ELISA, RT-PCR.
- No specific antiviral; supportive care (fluids, monitoring platelets).
- Vaccines: Dengvaxia (Sanofi, only for seropositive individuals); Qdenga (Takeda).
- Vector control is the mainstay of prevention.

Final Answer: DENV (a Flavivirus) transmitted by *Aedes* mosquito $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q28

Q29.

Solution

Concept — MHC — self-recognition and adaptive immunity: Major Histocompatibility Complex; in humans, also called HLA (Human Leukocyte Antigens). Genes located on chromosome 6. Among the most polymorphic loci in the human genome.

Step 1 — Two main classes:

- **MHC class I (HLA-A, -B, -C):** on essentially ALL nucleated cells (not erythrocytes, which are anucleate). Present **endogenous** (intracellular) peptides — normally derived from self-proteins broken down by the proteasome; in infection, viral proteins or in cancer, abnormal proteins. Bound peptides are short (8–10 aa). Recognised by $CD8^+$ cytotoxic T cells.
- **MHC class II (HLA-DR, -DP, -DQ):** on professional antigen-presenting cells (APCs): dendritic cells, macrophages, B cells. Present **exogenous** peptides (taken up by phagocytosis/endocytosis from extracellular pathogens). Bound peptides are longer (13–25 aa). Recognised by $CD4^+$ helper T cells.

Step 2 — The functional logic:

- MHC I broadcasts "what's inside the cell" $\rightarrow$ $CD8^+$ T cells kill infected cells.
- MHC II broadcasts "what's outside in the body" $\rightarrow$ $CD4^+$ helper T cells coordinate the broader immune response.
- This dichotomy ensures appropriate response to intracellular (viruses, intracellular bacteria) vs extracellular (most bacteria, parasites) pathogens.

Step 3 — MHC polymorphism and population-level immunity:

- Thousands of alleles per MHC locus across the human population; each individual is heterozygous.
- Heterozygosity allows wider peptide repertoire $\rightarrow$ better immune coverage.
- Pathogens that escape one HLA haplotype's presentation may be caught by another.
- Some HLA alleles are associated with autoimmune diseases (HLA-B27 $\rightarrow$ ankylosing spondylitis; HLA-DR3/4 $\rightarrow$ type 1 DM).

Step 4 — Transplant rejection:

- HLA mismatch is the major basis of allograft rejection.
- Donor and recipient must be HLA-matched (or rejection suppressed by immunosuppressants).
- Identical twin grafts (HLA-identical) are accepted without immunosuppression.
- Bone marrow transplant: needs even closer matching to prevent graft-vs-host disease (GVHD).

Step 5 — Nobel Prize: The discovery of MHC restriction in T-cell recognition (Doherty and Zinkernagel) earned the 1996 Medicine Nobel.

Final Answer: MHC presents peptide antigens to T-cell receptors $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q29

Q30.

Solution

Concept — Opioids — the most potent CNS depressants: Opium is the dried latex of unripe seed capsules of the opium poppy *Papaver somniferum*. Contains ~ 20 alkaloids, of which morphine (10–16%) and codeine (0.5–3%) are the main pharmacologically active ones.

Step 1 — Classification:

- **Natural opium alkaloids:** morphine, codeine, thebaine, papaverine.
- **Semi-synthetic:** heroin (diacetylmorphine, made from morphine), oxycodone, hydrocodone, buprenorphine.
- **Fully synthetic:** fentanyl, methadone, tramadol, pethidine. Fentanyl is $\sim 100\times$ more potent than morphine.

Step 2 — Mechanism — opioid receptors: Three main types — μ (mu), δ (delta), κ (kappa) — all G-protein-coupled receptors. Activation:

- Inhibits adenylate cyclase $\rightarrow$ lowers cAMP.
- Opens K^+ channels $\rightarrow$ hyperpolarises neurons.
- Closes Ca^{2+} channels $\rightarrow$ decreases neurotransmitter release.

Net effect: depresses neuronal firing, particularly in pain pathways, brainstem, limbic system.

Step 3 — Endogenous opioid system: The body produces its own opioid peptides: endorphins, enkephalins, dynorphins. They modulate pain perception, mood, stress responses, reward.

Step 4 — Acute effects:

- Powerful analgesia (clinically important for severe pain).
- Sedation, drowsiness.
- Euphoria, "rush" (especially with fast-acting IV heroin or smoked).
- Respiratory depression (the principal cause of overdose death — the brainstem stops responding to CO_2).
- Pupillary constriction (pinpoint pupils — a classic sign of opioid overdose).
- Constipation, nausea, vomiting.

Step 5 — Chronic use:

- Tolerance: increasing doses required.

- Physical dependence: withdrawal syndrome on cessation (yawning, lacrimation, rhinorrhoea, diarrhoea, sweating, dilated pupils, anxiety, severe craving).
- Psychological dependence: severe addiction.
- Increased risk of HIV/HCV (IV needle sharing), endocarditis, overdose.

Step 6 — Treatment:

- Acute overdose: naloxone (opioid receptor antagonist; reverses respiratory depression in minutes).
- Long-term: methadone or buprenorphine maintenance therapy; counselling; harm reduction.

Final Answer: CNS depressants acting on μ, δ, κ opioid receptors $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q30

Q31.

Solution

Concept — r-selection and K-selection: A life-history framework proposed by Robert MacArthur and E. O. Wilson (1967) to classify species by reproductive strategy.

Step 1 — The variables:

- r = intrinsic rate of natural increase (per capita growth rate).
- K = carrying capacity of the environment.
- Logistic growth: $\mathrm{dN/dt = rN(K - N)/K}$. When $N \ll K$, growth is r-dominated; when $N \to K$, growth is K-dominated.

Step 2 — r-selected species:

- Live in unstable, unpredictable environments where mortality is high and density-independent (storms, disturbance, predation).
- Strategy: maximise reproductive output before being killed.
- Traits:
  - Small body size.
  - Short life span; early maturity.
  - Many small offspring per reproductive event.
  - Little or no parental care.

  - Iteroparity (single reproductive event in many cases, or repeated very early in life).
  - High dispersal ability.
- Examples: most insects, weeds, dandelions, rodents, many fish (cod), bacteria, oysters.
- Population fluctuates wildly around K, often well below or well above.

Step 3 — K-selected species:

- Live in stable, predictable environments where competition matters more than disturbance.
- Strategy: produce high-quality, competitive offspring that can survive density-dependent pressures.
- Traits:
  - Large body size.
  - Long life span; late maturity.
  - Few large offspring per reproductive event.
  - Intensive parental care.
  - Repeated reproduction over long lifespan.
- Examples: elephants, whales, primates (including humans), oak trees, eagles, large carnivores.
- Population near carrying capacity (K), regulated by density-dependent factors (food, competition, disease).

Step 4 — A spectrum, not a dichotomy: Most species are intermediate; classification is relative. Modern alternatives include the slow-fast continuum (Pianka) and the C-S-R triangle for plants (Grime: Competitors, Stress-tolerators, Ruderals).

Step 5 — Conservation relevance: K-strategists are particularly vulnerable to extinction (slow reproduction, can't bounce back from population crash); r-strategists rebound quickly but often become invasive pests when introduced.

Final Answer: r-selected: short life, many offspring, little parental care, fluctuating populations $\Rightarrow$ C

Answer: (C) Go Back to Q31

Q32.

Solution

Concept — Ecological succession: The orderly, predictable change in species composition of a community over time, ending (in theory) in a stable climax community.

Step 1 — Two types:

- **Primary succession:** begins on previously sterile substrate, with NO pre-existing soil or biota. Examples: cooled lava flows, newly exposed glacial rock, sand dunes, the surface of new volcanic islands.
- **Secondary succession:** begins on disturbed but not sterilised substrate, where soil and some propagules remain. Examples: after fire, agricultural abandonment (old-field succession), landslide, hurricane. Faster than primary succession.

Step 2 — Classic example of primary succession — glacier retreat at Glacier Bay, Alaska:

(a) Bare rock and gravel left by retreating glacier (0 yr).
(b) Pioneer stage: lichens, cyanobacteria, mosses ($\sim$ 10–50 yr). They weather rock and add organic matter; some fix nitrogen.
(c) Herbaceous stage: *Dryas drummondii* (mountain avens, has nitrogen-fixing root symbionts) and other herbs ($\sim$ 50–80 yr).
(d) Shrub stage: alder (also N-fixing through actinorhizal symbiosis) thickets ($\sim$ 80–120 yr).
(e) Sitka spruce forest ($\sim$ 200–300 yr).
(f) Climax: spruce-hemlock forest ($\sim$ 700+ yr).

Step 3 — The seral stages: The intermediate stages are called **seres** (or seral communities); each modifies the environment in ways that favour the next sere (facilitation).

Step 4 — Mechanisms (Connell-Slatyer):

- Facilitation: early species modify environment, making it suitable for later species.
- Tolerance: later species can grow in conditions early ones created, regardless of facilitation.
- Inhibition: early species can inhibit later ones until disturbance creates an opening.

Step 5 — Climax community: Self-perpetuating, stable, in equilibrium with prevailing climate; species composition relatively constant; energy capture and respiration balanced (net production approaches zero).

Final Answer: Primary succession (on previously sterile substrate) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q32

Q33.

Solution

Concept — CRISPR-Cas9 — the genome-editing revolution: Discovery awarded the 2020 Nobel Prize in Chemistry to Emmanuelle Charpentier and Jennifer Doudna (their seminal 2012 paper). The most transformative tool in biology of the 21st century so far.

Step 1 — Bacterial origin: CRISPR = "Clustered Regularly Interspaced Short Palindromic Repeats". An adaptive immune system of bacteria and archaea protecting against phages. Bacteria capture short fragments of invading phage DNA, store them as "spacers" in their genome, transcribe them as guide RNAs, and use Cas (CRISPR-associated) proteins to cleave matching phage DNA on re-encounter.

Step 2 — The engineered tool: Charpentier and Doudna re-engineered the type II CRISPR-Cas9 system into a programmable nuclease for genome editing:

- **Guide RNA (gRNA, ~ 100 nt):** programmable; the $5'$ end has a ~ 20-nt sequence complementary to the target DNA.
- **Cas9 protein:** an endonuclease (RNA-guided) that cleaves both DNA strands creating a blunt-ended double-strand break (DSB).
- Together, gRNA + Cas9 form a ribonucleoprotein that scans the genome, finds the matching sequence (adjacent to a PAM motif, NGG for SpCas9), and cuts.

Step 3 — The cellular repair fills in:

- **NHEJ (non-homologous end joining):** error-prone; introduces small insertions/deletions (indels) at the cut site. Used for gene knockout (frameshift disrupts the protein).
- **HDR (homology-directed repair):** precise; if a donor DNA template with homology arms is supplied, the cell repairs the break using it $\rightarrow$ allows precise edits, insertions, replacements.

Step 4 — Applications:

- Loss-of-function genetic screens (whole-genome libraries of gRNAs).
- Disease modelling (creating cell lines / mice with disease-causing mutations).
- Crop improvement (CRISPR-edited tomatoes, rice, wheat with improved traits).
- Therapeutic gene editing: Casgevy (2023, first CRISPR drug approved by FDA) for sickle cell disease and β-thalassaemia — modifies BCL11A to reactivate foetal haemoglobin in patients' haematopoietic stem cells.
- Base editing, prime editing: refinements that introduce specific point mutations without double-strand breaks.

Step 5 — Ethical considerations: The 2018 He Jiankui scandal (germline editing of human embryos for HIV resistance) provoked international condemnation. Current consensus: somatic edits OK for therapy; germline edits banned in most jurisdictions.

Final Answer: gRNA-Cas9 creates targeted double-strand break $\Rightarrow$ B

Answer: (B) Go Back to Q33

Q34.

Solution

Concept — Golden Rice — biofortification by transgenesis: Developed by Ingo Potrykus and Peter Beyer (2000). The flagship example of using transgenic crops to address malnutrition.

Step 1 — The problem: Vitamin A deficiency affects 250 million children worldwide; causes 250,000–500,000 cases of irreversible blindness annually; $\sim 1-2$ million deaths (mostly children) due to increased infection susceptibility. Most acute in populations where polished white rice is the staple.

Step 2 — The biology: White rice endosperm contains no β-carotene because the biosynthetic pathway is not active in this tissue (though the leaves have it). Vitamin A is synthesised in the body from dietary β-carotene (pro-vitamin A).

Step 3 — The genetic engineering: Two transgenes inserted into rice endosperm-specific expression cassette:

- *psy* (phytoene synthase): originally from daffodil; later version uses maize *psy* for higher activity. Converts the precursor geranylgeranyl pyrophosphate (GGPP) to phytoene.
- *crtI* (carotene desaturase): from the soil bacterium *Erwinia uredovora*. Con-

verts phytoene through several desaturations to lycopene, which the endogenous rice cyclase converts to β-carotene.

Result: rice grains have a yellow/orange colour ($\rightarrow$ "golden"); contain $\sim 30\,\mu g$ β-carotene/g rice in Golden Rice 2 (the improved version).

Step 4 — Deployment:

- Approved as safe by FAO, WHO, FDA, etc.
- In 2018 approved in USA, Canada, Australia, NZ.
- 2021: Philippines became the first country to approve commercial cultivation.
- Bangladesh approved in 2023.

Step 5 — Controversy: Long delayed by opposition from anti-GM advocacy groups, despite scientific consensus on safety. Some scientists view this as one of the most consequential delays in public health history.

Final Answer: Daffodil/maize *psy* + *Erwinia crtI* for β-carotene in endosperm $\Rightarrow$ C

Answer: (C) Go Back to Q34

Q35.

Solution

Concept — Influenza antigenic variation: The remarkable ability of influenza A virus to evade pre-existing immunity, leading to annual epidemics and occasional pandemics.

Step 1 — Influenza A biology:

- Family Orthomyxoviridae.
- Enveloped, negative-sense, single-stranded RNA virus.
- Genome is segmented into 8 separate RNA pieces (key to reassortment).
- Subtyped by the two surface glycoproteins: **H**aemagglutinin (binds sialic acid on host cell surface; 18 subtypes H1–H18) and **N**euraminidase (cleaves sialic acid to release new virions; 11 subtypes N1–N11).
- Current human seasonal strains: H1N1, H3N2, plus influenza B.

Step 2 — Antigenic drift:

- Gradual accumulation of point mutations in the HA and NA genes (RNA polymerase lacks proofreading).

- Slight changes in surface antigens; some escape antibodies from previous infection/vaccination.
- Cause of **annual seasonal flu epidemics**; necessitates annual reformulation of the flu vaccine.

Step 3 — Antigenic shift:

- Sudden, dramatic change due to **reassortment** of the 8 RNA segments when two different influenza A strains infect the same host cell.
- "Mixing vessel" is often a pig (susceptible to both human and avian flu) or a bird.
- Produces a novel HA-NA combination to which the human population has no prior immunity $\rightarrow$ **pandemic potential**.
- Examples: 1918 Spanish flu (H1N1, ≥ 50 million deaths); 1957 Asian (H2N2); 1968 Hong Kong (H3N2); 2009 swine flu (H1N1pdm09).

Step 4 — Pandemic risk: Highly pathogenic avian influenza (HPAI) strains like H5N1 and H7N9 are watched closely; currently have high case-fatality rate but poor human-to-human transmission. A reassortment that combined a deadly avian HA with efficient human transmissibility would be catastrophic.

Step 5 — Vaccines and antivirals:

- Annual trivalent or quadrivalent vaccines (egg-grown or cell-cultured).
- Antivirals: neuraminidase inhibitors (oseltamivir, zanamivir); polymerase inhibitors (baloxavir).
- Universal flu vaccine (targeting conserved HA stem) under development.

Final Answer: Antigenic drift (point mutations) + antigenic shift (reassortment) $\Rightarrow \boxed{\text{D}}$

Answer: (D) Go Back to Q35

Q36.

Solution

Concept — Type-I (IgE-mediated, immediate) hypersensitivity: The classical "allergic reaction" — hay fever, asthma, food allergy, anaphylaxis. Pathway elucidated by Coombs and Gell (1963).

Step 1 — Sensitisation phase: First exposure to allergen (pollen, peanut, dust mite). Allergen-specific B cells class-switch to produce IgE antibodies (driven by Th2 cells, IL-4). IgE binds with very high affinity to FcϵRI receptors on mast cells (in tissues) and basophils (in blood). Person is now "sensitised"; usually no symptoms yet.

Step 2 — Effector phase (on re-exposure):

- Allergen crosslinks two or more IgE molecules bound on mast cell surface.
- Signal transduction $\rightarrow$ Ca^{2+} influx $\rightarrow$ rapid mast cell degranulation (within seconds).
- Pre-formed mediators released from granules:
  - **Histamine** (the dominant mediator) — vasodilation, increased capillary permeability (causing oedema and the wheal-and-flare reaction), bronchoconstriction, glandular secretion, sensory nerve activation (itching).
  - Tryptase, chymase, heparin.
- Newly synthesised lipid mediators (minutes): leukotrienes (LTC_4, LTD_4, LTE_4 — "slow-reacting substance of anaphylaxis"), prostaglandin D_2.
- Cytokines (hours): IL-4, IL-5, TNFα — recruit eosinophils, sustain allergic inflammation (late phase).

Step 3 — Clinical manifestations:

- Local (e.g. allergic rhinitis): sneezing, runny nose, itchy eyes.
- Asthma: bronchoconstriction, mucus, wheezing.
- Urticaria (hives), atopic dermatitis.
- Food allergies: GI symptoms; can progress to anaphylaxis.
- **Anaphylaxis:** systemic, life-threatening — airway oedema, bronchospasm, circulatory collapse, hypotension. Death within minutes if untreated.

Step 4 — Treatment:

- **Antihistamines** (H_1-receptor blockers): cetirizine, loratadine, fexofenadine — for mild-moderate allergy.

- Glucocorticoids: for inflammation control.
- Leukotriene receptor antagonists: montelukast — for asthma.
- Mast-cell stabilisers: cromolyn sodium.
- **Anaphylaxis:** adrenaline (epinephrine) IM — the only life-saving intervention; reverses vasodilation and bronchoconstriction within minutes. Patients with known anaphylaxis carry auto-injectors (EpiPen).
- Immunotherapy (allergy shots, sublingual immunotherapy): induces immune tolerance over years.
- Anti-IgE monoclonal antibody (omalizumab) for severe allergic asthma.

Final Answer: Histamine, the principal preformed mediator $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q36

Q37.

Solution

Concept — Ecological pyramids: Three classical ways to represent trophic structure of an ecosystem (Eltonian pyramids).

Step 1 — Three types:

- **Pyramid of numbers:** number of organisms at each trophic level. Can be inverted (e.g. one tree supports thousands of insects, that support a few birds: tree-tip narrow but middle wide).
- **Pyramid of biomass:** total dry mass of organisms at each level. Can also be inverted (classic ocean example: small standing crop of fast-turnover phytoplankton supports a much larger standing crop of zooplankton at any given moment).
- **Pyramid of energy:** rate of energy flow ($\text{kJ m}^{-2}\,\text{yr}^{-1}$) through each trophic level. **Always upright**.

Step 2 — Why energy pyramid is always upright: Lindemann's $\sim 10\%$ rule (1942): only about 10% of the energy in one trophic level is transferred to the next; the remaining $\sim 90\%$ is lost to:

- Respiration (heat lost to the environment per Second Law of thermodynamics).
- Undigested material (passed in faeces).
- Heat from inefficient biochemical conversions.

Therefore each successive trophic level contains an order of magnitude less energy flow than the one below it. There is no thermodynamic way to invert this.

Step 3 — Numerical example (Silver Springs, FL — Howard Odum, 1957):

Trophic level	kcal m^{-2} yr^{-1}
Producers (plants)	20,810
Herbivores	3,368
Carnivores	383
Top carnivores	21

Note the dramatic loss between successive levels — a clear pyramidal pattern.

Step 4 — Implications:

- Food chains are typically only 3–5 trophic levels long; energy runs out.
- Top carnivores have small populations and large territory requirements; vulnerable to extinction.
- Eating lower on the food chain (vegetarianism) is energetically more efficient — supports more people per area of land.
- Energy flow is one-way (unlike nutrient cycles, which loop).

Final Answer: Only $\sim 10\%$ energy transferred per level; unidirectional flow $\Rightarrow$ C

Answer: (C) Go Back to Q37

Q38.

Solution

Concept — Climax community: The endpoint of ecological succession — a stable, self-perpetuating community in equilibrium with the prevailing climate, soil, and disturbance regime.

Step 1 — Characteristics:

- Composed of dominant species adapted to long-term local conditions.
- Stable species composition over time (in absence of disturbance).
- Maximum biodiversity and niche specialisation for the region.
- Net primary production $\approx$ ecosystem respiration $\Rightarrow$ near-zero net carbon storage; standing biomass at its peak.
- Detritivore and decomposer communities highly developed.
- Self-regulating; resistant to most small disturbances.

Step 2 — Examples (region-dependent):

- Eastern temperate North America: oak-hickory deciduous forest.

- Boreal regions: spruce-fir taiga.
- Tropical wet equatorial: tropical rainforest.
- Arid regions: desert scrub.
- Mediterranean: chaparral.
- Tropical grasslands: savanna.

Step 3 — The mono-climax vs poly-climax debate:

- Frederic Clements (1916, mono-climax theory): only one true climax per climatic region (determined by climate alone).
- Henry Gleason and others (poly-climax theory): multiple climaxes possible within a region, depending on soil, drainage, topography, disturbance.
- Modern view: climaxes are dynamic mosaics shaped by climate, soils, and recurrent disturbance (fire, herbivory, storms, human activity).

Step 4 — Disturbance and the patch-dynamics view: Real ecosystems are mosaics of patches at different successional stages, constantly cycled by small-scale disturbance (treefalls, fire). Strict equilibrium is rare; the concept of climax remains useful as a reference state.

Final Answer: Climax community $\Rightarrow$ D

Answer: (D) Go Back to Q38

Q39.

Solution

Concept — Gibberellins (GAs): A large family of > 130 structurally related tetracyclic diterpenoid plant hormones (only a few biologically active: GA_1, GA_3, GA_4, GA_7).

Step 1 — Discovery: First isolated from the fungal pathogen *Gibberella fujikuroi* (now *Fusarium fujikuroi*), the cause of "bakanae" ("foolish seedling") disease of rice — infected seedlings grow extremely tall and spindly, then collapse. Japanese scientists Eiichi Kurosawa (1926) traced this to a soluble fungal product; later named gibberellin.

Step 2 — Major physiological effects:

- **Stem elongation:** promotes internode elongation through cell elongation and cell division at the subapical meristem. In some plants, dramatic "bolting" in rosette plants (cabbage, lettuce) when day length triggers GA production.

- **Seed germination:** breaks seed dormancy. In barley malting, GA from the embryo activates the aleurone layer to synthesise α-amylase, which mobilises starch reserves to feed the growing embryo. This is exploited commercially: in beer-making, exogenous GA can accelerate malt production.
- **Flowering:** promotes flowering in some plants, especially long-day plants and those requiring vernalisation (substitutes for cold treatment in some species).
- **Sex expression:** promotes male flower development in some monoecious cucurbits.
- **Fruit growth and parthenocarpy:** GA_3 sprays produce larger, looser bunches of seedless grapes (Thompson seedless).

Step 3 — Biosynthesis: From geranylgeranyl pyrophosphate (GGPP) via *ent*-kaurene in plastids, then ER and cytosol. Regulated by KAO, GA20ox, GA3ox enzymes. Inactivation by GA2ox.

Step 4 — Signal transduction: GA binds the GID1 receptor $\rightarrow$ recruits DELLA repressor proteins $\rightarrow$ DELLA is ubiquitinated and degraded by the proteasome $\rightarrow$ growth-promoting transcription factors are released to act.

Step 5 — Practical and breeding importance:

- "Green Revolution" dwarf wheat and rice varieties (semi-dwarfs developed by Norman Borlaug and others) carry mutations that reduce GA response or biosynthesis $\rightarrow$ shorter, stockier plants that resist lodging and respond to fertilizer with higher grain yields. This single trait fed billions.
- GA inhibitors (paclobutrazol, daminozide) used commercially to dwarf ornamentals.
- Brewing industry: GA application to barley enhances malt production.

Final Answer: Break dormancy, promote bolting/elongation, α-amylase induction $\Rightarrow \boxed{\text{A}}$

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

Q40.

Solution

Concept — Naming convention for restriction enzymes: Standardised by Smith and Nathans (1973). Reflects the source organism systematically. Restriction endonucleases recognise specific short DNA sequences (typically 4–8 bp palindromes) and cleave both strands.

Step 1 — The four-part name: Example: ***EcoRI***

- *E* (italic, capitalised): first letter of the genus = *Escherichia*.
- *co* (italic, lowercase): first two letters of the species name = *coli*.
- R (roman, capital): the strain identifier = strain RY13.
- I (roman, Roman numeral): the order of identification of this enzyme from this strain = the first.

Italics for the genus and species reflect biological nomenclature convention.

Step 2 — Other examples:

- *Hin*dIII: from *Haemophilus influenzae* serotype d, the third enzyme isolated.
- *Bam*HI: from *Bacillus amyloliquefaciens* strain H, first enzyme.
- *Sma*I: from *Serratia marcescens*, first enzyme.
- *Pst*I: from *Providencia stuartii*.
- *Not*I: from *Nocardia otitidis-caviarum*.

Step 3 — Recognition sites and cuts: Common type-II restriction enzymes recognise palindromic sequences (sequence reads the same 5' to 3' on both strands):

- *EcoRI*: $G^{\downarrow}AATTC$ — leaves 5′ AATT sticky overhangs.
- *Hin*dIII: $A^{\downarrow}AGCTT$ — leaves 5′ AGCT sticky overhangs.
- *Bam*HI: $G^{\downarrow}GATCC$ — leaves 5′ GATC sticky overhangs.
- *Sma*I: $CCC^{\downarrow}GGG$ — blunt cut.
- *Pst*I: $CTGCA^{\downarrow}G$ — 3′ overhangs.

Step 4 — Why bacteria have restriction enzymes: A primitive immune system against bacteriophage and foreign DNA. Bacteria methylate their own DNA at the recognition sites (*modification*), so their own enzymes do not cut; foreign DNA (unmethylated) is cleaved. Restriction-modification systems.

Step 5 — Importance in molecular biology: Restriction enzymes revolutionised molecular biology (Werner Arber, Hamilton Smith, Daniel Nathans — 1978 Nobel Prize): made it possible to cut DNA reproducibly at defined sites, the foundation of

all DNA cloning. Modern collections feature $> 3{,}500$ type II enzymes with various specificities (REBASE database).

Final Answer: *E*scherichia *c*oli *R*Y13 first enzyme $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) **Go Back to Q40**

Answer Key

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