BITSAT Biology Sample Paper – 9

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. The large central vacuole of a mature plant cell (often $> 80\%$ of the cell volume) is bounded by a single membrane called the:

(A) Plasmalemma (this is the plasma membrane)

(B) Endoplasmic reticulum

(C) Tonoplast — which actively pumps H^+ (via V-ATPase and pyrophosphatase) into the vacuolar lumen, generating an acidic pH and driving secondary transport of ions, sugars and pigments (anthocyanins); also stores water (providing turgor), nutrients, defensive secondary metabolites and waste products

(D) Plasmodesma

Q2. The eukaryotic plasma membrane shows marked transbilayer lipid asymmetry. In a resting RBC for example:

(A) The two leaflets are randomly mixed

(B) Both leaflets contain only phosphatidylcholine

(C) There is no asymmetry

(D) Outer (exoplasmic) leaflet is enriched in phosphatidylcholine and sphingomyelin (choline-containing lipids); inner (cytoplasmic) leaflet is enriched in phosphatidylethanolamine, phosphatidylserine and phosphatidylinositol; maintained by flippases (ATP-driven) and scramblases (during apoptosis, exposing PS as an "eat-me" signal)

Q3. The enzyme telomerase (Carol Greider, Elizabeth Blackburn, Jack Szostak; Nobel 2009) solves the end-replication problem by:

(A) Cutting telomeres shorter

(B) Using its own intrinsic RNA template (TERC component) to add tandem repeats of TTAGGG (in vertebrates) to the $3'$ overhang of chromosome ends, lengthening telomeres against the shortening that occurs each cell division

(C) Replacing telomeres with centromeric DNA

(D) Inserting introns into the telomeric region

Q4. In eukaryotic mRNA maturation, the precise removal of introns and joining of exons is carried out by a large RNA-protein complex called the:

(A) Ribosome (this performs translation)

(B) Spliceosome — assembled at each intron from 5 small nuclear ribonucleoproteins (snRNPs: U1, U2, U4, U5, U6) and ~ 200 accessory proteins; recognises $5'$ splice donor, branch-point adenosine, $3'$ splice acceptor; catalyses lariat formation and exon ligation

(C) Nucleolus

(D) Proteasome

Q5. Proteins destined for secretion, lysosomes, or the plasma membrane in eukaryotic cells are co-translationally targeted to the ER by an N-terminal:

(A) Nuclear localisation signal (this targets proteins to the nucleus)

(B) Mitochondrial targeting peptide (this targets to mitochondria)

(C) Peroxisomal targeting signal (PTS1, PTS2)

(D) Signal peptide (15–30 hydrophobic amino acids) — recognised by the signal recognition particle (SRP) as it emerges from the ribosome; SRP halts translation and docks the ribosome on the ER membrane via SRP receptor; protein is then co-translationally translocated through the Sec61 translocon; signal peptide is cleaved by signal peptidase

Q6. Very-long-chain fatty acids (VLCFAs, $\geq C22$) and branched-chain fatty acids undergo β-oxidation primarily in:

(A) Peroxisomes — initial cycles shorten the chain to medium length; further shortening in mitochondria. First peroxisomal step uses fatty-acyl-CoA oxidase (electrons go directly to O_2, giving H_2O_2, not $FADH_2$). Defects cause X-linked adrenoleukodystrophy and Zellweger syndrome

(B) Lysosomes

(C) Cytosol

(D) Nucleus

Q7. Entry of a cell into mitosis (the G_2/M transition) is driven by the activation of:

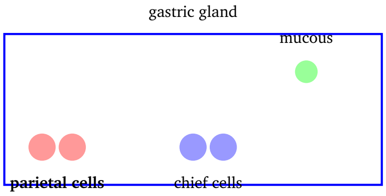
(A) p53 tumour suppressor

(B) M-phase promoting factor (MPF) — a heterodimer of cyclin B and cyclin-dependent kinase 1 (CDK1/CDC2); accumulates in G_2, becomes activated by cdc25 phosphatase, and phosphorylates nuclear lamins (envelope breakdown), histone H1 (chromatin condensation), and other substrates to drive mitotic entry

(C) Telomerase

(D) Insulin

Q8. In the gastric (stomach) mucosa shown below, the cells that secrete hydrochloric acid (HCl) and intrinsic factor are:



(A) Chief (zymogen) cells (secrete pepsinogen and gastric lipase, NOT HCl)

(B) Mucous neck cells (secrete protective mucus)

(C) Parietal (oxyntic) cells — in the upper gastric glands; pump H^+ via the H^+/K^+-ATPase (proton pump), Cl^- following; HCl creates pH ~ 1–2, activating pepsinogen and killing pathogens; also secrete intrinsic factor for ileal vitamin B_{12} absorption

(D) Enteroendocrine cells

Q9. Premature infants (born < 28 weeks gestation) frequently develop infant respiratory distress syndrome (IRDS) because their alveoli collapse on each expiration. The molecule whose insufficient production causes this is:

(A) Haemoglobin

(B) Mucus from goblet cells

(C) Ciliary fluid

(D) Pulmonary surfactant (a mixture of dipalmitoylphosphatidylcholine, surfactant proteins SP-A/B/C/D, cholesterol) secreted by Type II pneumocytes; reduces alveolar surface tension by Laplace mechanism, preventing alveolar collapse at end-expiration; secretion begins ~ 24 weeks gestation and rises rapidly ~ 35 weeks; antenatal corticosteroids accelerate maturation

Q10. Cardiac output (CO), the volume of blood pumped by the heart per minute, equals:

(A) Stroke volume (SV) $\times$ heart rate (HR). At rest in adults: SV $\sim$

$70\,\text{mL/beat} \times \text{HR} \sim 72\,\text{beats/min} \approx 5\,\text{L/min}$. During maximal exercise, CO can rise 5–7 fold via increases in both HR and SV

(B) Stroke volume + heart rate (sum)

(C) Diastolic blood pressure × pulse rate

(D) Systolic blood pressure alone

Q11. Atrial natriuretic peptide (ANP), released from cardiac atrial myocytes in response to atrial stretch (volume overload), acts on the kidney to:

(A) Promote Na^+ retention and water reabsorption

(B) Increase Na^+ excretion (natriuresis) and water loss (diuresis) by promoting GFR (afferent dilation, efferent constriction) and inhibiting Na^+ reabsorption in the collecting duct; opposes the renin-angiotensin-aldosterone system; lowers blood pressure and blood volume

(C) Stimulate ADH release from the posterior pituitary

(D) Cause vasoconstriction

Q12. The brain region primarily responsible for coordination of voluntary movement, balance and fine motor control — containing $> 50\%$ of all the neurons in the brain despite occupying only $\sim 10\%$ of its volume — is the:

(A) Cerebrum (responsible for cognition, sensation, voluntary motor planning, language)

(B) Medulla oblongata (controls cardiorespiratory centres)

(C) Cerebellum — "little brain"; located behind the brainstem; receives copies of motor commands and proprioceptive feedback; computes corrections to make movements smooth and accurate. Damage causes ataxia, intention tremor, dysmetria

(D) Hypothalamus (homeostasis, neuroendocrine, autonomic, drives)

Q13. Aldosterone, the principal mineralocorticoid, acts on the distal convoluted tubule and cortical collecting duct of the kidney to:

(A) Increase Na^+ reabsorption (and water following) and K^+ excretion — by upregulating ENaC sodium channels and Na/K-ATPase; opposes ANP; raises blood volume and pressure. Stimulated by angiotensin II and high plasma K^+

(B) Decrease Na^+ reabsorption and retain K^+

(C) Excrete glucose

(D) Excrete water without affecting salt

Q14. Leaf abscission (the controlled shedding of leaves) involves a specialised abscission zone at the base of the petiole. The hormone that initiates abscission and the one that retards it are, respectively:

(A) Auxin promotes, cytokinin inhibits

(B) GA promotes, ABA inhibits

(C) Ethylene (promotes abscission — activates cellulases and polygalacturonases in the abscission zone) versus auxin (delays abscission as long as the leaf is producing auxin; abscission begins when auxin levels fall in ageing leaves)

(D) Cytokinin promotes, GA inhibits

Q15. The Bombay phenotype (discovered in Bombay, India in 1952) is a rare blood group case in which individuals with genotypes I^AI^A, I^AI^B, etc. nevertheless type as "O". This illustrates:

(A) Incomplete dominance at the ABO locus

(B) Codominance of A and B

(C) Multiple alleles at a single locus

(D) Recessive epistasis: the unrelated H locus encodes the H precursor antigen, on which A and B antigens are built. Bombay individuals are homozygous for the recessive h allele (genotype hh), produce no H antigen, so neither A nor B can be displayed regardless of their ABO genotype; they appear O but their serum has anti-H antibodies and they can only receive blood from other Bombay donors

Q16. Francis Crick's "central dogma" of molecular biology (1958) states that genetic information flows:

(A) DNA → RNA → Protein — the unidirectional flow of sequence information from genome to functional protein, via transcription and translation. Reverse transcription (RNA → DNA, in retroviruses) and RNA replication were later additions; protein → DNA/RNA flow has never been observed

(B) Protein → RNA → DNA

(C) RNA → Protein only

(D) DNA → Protein directly (no RNA)

Q17. During the elongation cycle of protein synthesis, the ribosome has three tRNA binding sites. The incoming aminoacyl-tRNA enters the:

(A) P (peptidyl) site

(B) A (aminoacyl) site — where the new aminoacyl-tRNA enters, codon-paired with the mRNA; the peptidyl transferase center then transfers the growing peptide from the P-site tRNA onto the A-site amino acid; translocation moves A → P, P → E (exit), and exposes a new codon at A

(C) E (exit) site

(D) Stop codon

Q18. When a small group of individuals breaks off from a large parent population to colonise a new area, the new population's allele frequencies are usually different from the parent population. This non-selective evolutionary mechanism is called the:

(A) Bottleneck effect (size collapse of an existing population, not formation of a new one)

(B) Genetic drift in general

(C) Founder effect — a special case of genetic drift; the small founding group is by chance not a representative sample of the parent gene pool. Examples: high prevalence of Ellis-van Creveld syndrome among the Pennsylvania Amish; Huntington's disease in Lake Maracaibo (Venezuela); high prevalence of porphyria variegata among South African Afrikaners (traced to a single 17th-century Dutch immigrant)

(D) Natural selection

Q19. The Biological Species Concept (BSC; Ernst Mayr, 1942) defines a species as:

(A) Any group of organisms that look alike

(B) A group sharing a common name in the local language

(C) Organisms occupying the same geographical area

(D) A group of actually or potentially interbreeding natural populations that are reproductively isolated from other such groups — with the populations producing fertile offspring under natural conditions; emphasises reproductive isolation as the operational criterion of species boundaries

Q20. In humans, sex is determined by the presence or absence of the Y chromosome. The key gene on the Y chromosome that triggers male development is:

(A) SRY (Sex-determining Region Y) — encodes the Testis-Determining Factor, a transcription factor that diverts the bipotential gonad to develop as testis (instead of the default ovary) at ~ 7 weeks of gestation; testes then produce testosterone and anti-Müllerian hormone to mascularise the embryo

(B) TDF on the X chromosome

(C) BRCA1 on chromosome 17

(D) p53 on chromosome 17

Q21. Approximately 3–4 days after fertilisation in humans, the embryo consists of a solid mulberry-like ball of ~ 16 cells called the:

(A) Zygote

(B) Morula — still surrounded by the zona pellucida; cells beginning to compact (E-cadherin-mediated) and polarise; subsequent cavitation produces the blastocyst

(C) Gastrula

(D) Neurula

Q22. Gastrulation, the morphogenetic process during the 3rd week of human embryonic development, establishes:

(A) The single-cell zygote stage

(B) The trophoblast layer of the blastocyst

(C) The three primary germ layers (ectoderm, mesoderm, endoderm) by ingression of epiblast cells through the primitive streak, with the body axis (cranial-caudal, dorsal-ventral, left-right) established. Lewis Wolpert: "It is not birth, marriage, or death, but gastrulation which is truly the most important time in your life"

(D) The cleavage divisions

Q23. The class of immunoglobulin that crosses the placenta to confer passive immunity from mother to foetus is:

(A) IgM (the largest antibody, pentameric — cannot cross the placenta)

(B) IgA (secreted in mucosal sites and breast milk; not in placenta)

(C) IgE (mediates allergy; very low concentration; does not cross placenta)

(D) IgG — the only immunoglobulin class actively transported across the placenta (by neonatal Fc receptor, FcRn, on syncytiotrophoblast); confers passive immunity to the foetus and newborn for the first ~ 6 months; protects against many infections until the infant's own immune system matures

Q24. Medical Termination of Pregnancy (MTP) in India is legally permitted under the MTP Act (1971, amended 2021):

(A) Up to 20 weeks of gestation with the opinion of one registered medical practitioner, and up to 24 weeks for certain categories (rape survivors, minors, women with disability, foetal abnormality) with the opinion of two RMPs. After 24 weeks, only on the recommendation of a Medical Board in case of substantial foetal abnormalities

(B) Only up to 4 weeks

(C) At any stage of pregnancy without any medical opinion

(D) Only after 30 weeks

Q25. From outside to inside, the four wall layers of a typical anther are:

(A) Tapetum, middle layers, endothecium, epidermis

(B) **Epidermis** $\rightarrow$ **endothecium** $\rightarrow$ **middle layer(s)** $\rightarrow$ **tapetum** — only the tapetum is in direct contact with the developing microspores and provides their nourishment; the endothecium develops fibrous thickenings that drive dehiscence

(C) Endothecium, epidermis, tapetum, middle layers

(D) There is only one layer (the tapetum)

Q26. Megasporogenesis in flowering plants is the process by which:

(A) Microspores (pollen) are formed from microspore mother cells in the anther

(B) Pollen tubes germinate on the stigma

(C) A diploid megaspore mother cell (megasporocyte) inside the ovule undergoes meiosis to produce four haploid megaspores; usually three degenerate and only one (typically the chalazal) functional megaspore survives to give rise to the female gametophyte (embryo sac)

(D) Endosperm is formed

Q27. In some species (rice, sunflower, lettuce), seed dormancy is broken by which gaseous plant hormone?

(A) Abscisic acid

(B) Auxin

(C) Methylene

(D) Ethylene — promotes seed germination in many species (especially under flooded/anoxic conditions in rice), induces α-amylase activity in some, and breaks light-required dormancy in lettuce; produced by the seed itself or applied externally as ethephon

Q28. Charles Darwin (with his son Francis) showed in 1880 that the bending of grass coleoptiles toward light is governed by a signal originating from the tip. The hormone subsequently shown to mediate this is:

(A) Auxin (indole-3-acetic acid, IAA) — redistributed by phototropin photoreceptors to the shaded side; promotes cell elongation there; shoot bends toward light. The Cholodny-Went hypothesis (1926). PIN auxin-efflux carriers relocate to the lower/shaded side

(B) Cytokinin

(C) Gibberellin

(D) Ethylene

Q29. Red algae (Rhodophyta) such as *Porphyra* (nori, used to wrap sushi), *Gracilaria* (source of agar) and *Gelidium* are characterised by:

(A) Chlorophyll b in addition to a, and motile flagellated gametes

(B) Chlorophyll a + phycoerythrin (red) and phycocyanin (blue) accessory pigments; no flagellated stages anywhere in the life cycle (unique among algae); storage product is floridean starch; cell walls contain agar (yields agarose) or carrageenan

(C) Chlorophyll a + fucoxanthin (this is brown algae)

(D) Lack of any photosynthetic pigments

Q30. Phylum Annelida (earthworms, leeches, polychaete worms) is distinguished from other worm phyla by:

(A) Pseudocoel (this characterises Nematoda)

(B) Acoelomate body (this characterises Platyhelminthes)

(C) True coelom and **metameric segmentation** — body divided into a longitudinal series of similar segments (metameres) separated internally by septa; each segment with its own pair of nephridia (excretion), ganglia, gonads (in some) and longitudinal/circular muscles; closed circulation; setae for locomotion (in Oligochaeta and Polychaeta)

(D) Stinging cells

Q31. Class Aves (birds) is uniquely characterised by:

(A) Mammary glands and hair

(B) Naked seeds

(C) Cold-blooded ectothermy

(D) Feathers (made of keratin; modified epidermal scales — found in no other living animal class), forelimbs modified into wings, hollow pneumatic bones, four-chambered heart, endothermy, oviparous (laying calcified eggs with extra-embryonic membranes), and unique parabronchial lungs with through-flow ventilation (more efficient than mammalian tidal breathing)

Q32. Cholera, a severe diarrhoeal disease characterised by "rice-water" stools and rapid dehydration that can kill within hours, is caused by:

(A) *Vibrio cholerae* — a curved gram-negative rod (often biotypes El Tor or Classical, serogroups O1 and O139); produces cholera toxin (CT), an AB_5 toxin that ADP-ribosylates the $G\alpha_s$ subunit, locking adenylate cyclase in the "on" state in intestinal epithelial cells; the resulting massive efflux of Cl^- and water causes profuse watery diarrhoea

(B) *Salmonella typhi*

(C) *Plasmodium falciparum*

(D) *Mycobacterium leprae*

Q33. The complement system is a collection of ~ 30 plasma proteins that amplify and integrate innate and adaptive immune responses. It can be activated by three pathways. The terminal common product is:

(A) Antibody IgG

(B) Membrane Attack Complex (MAC, C5b-6-7-8-9_n) — assembles a pore in the pathogen membrane causing osmotic lysis; complement also opsonises pathogens (C3b for phagocyte recognition) and recruits inflammatory cells via anaphylatoxins (C3a, C5a)

(C) Interleukin-2

(D) Histamine

Q34. The two-step hepatic catabolism of ethanol involves:

(A) Direct excretion of ethanol in urine

(B) Conversion to glucose

(C) Alcohol dehydrogenase (ADH, NAD^+-dependent) oxidises ethanol to acetaldehyde, then aldehyde dehydrogenase (ALDH2, mitochondrial) oxidises acetaldehyde to acetate. Many East Asians carry a low-activity ALDH2 variant $\rightarrow$ acetaldehyde accumulates $\rightarrow$ "Asian flush"

(D) Conversion to glycerol

Q35. In ecology, the **fundamental niche** of a species is the full range of physical and biological conditions under which it could persist in the absence of competitors and predators; the **realised niche** is:

(A) Always identical to the fundamental niche

(B) Only the temperature range tolerated

(C) Just the food sources used

(D) The actually occupied subset of the fundamental niche, constrained by interspecific competition, predation, parasitism and other biotic interactions; classically demonstrated by Connell's barnacle experiment (1961) where *Chthamalus* (fundamental niche extending into lower intertidal) is restricted to the upper intertidal by competition with *Balanus*

Q36. Real ecosystems are better represented by food **webs** rather than simple linear food chains because:

(A) Most consumers are omnivorous and feed on multiple species at different trophic levels; many species are eaten by several different predators. The web has many cross-connections (trophic links) representing this complexity. Webs are more stable against perturbations than linear chains because alternative paths exist

(B) All animals eat only one type of food

(C) Plants are the only food source

(D) Decomposers have no role

Q37. The classic technique developed by Edwin Southern (1975) for detecting a specific DNA sequence among many fragments is:

(A) Northern blot (for RNA)

(B) **Southern blot** — DNA is digested with restriction enzymes, separated by agarose gel electrophoresis, denatured, transferred ("blotted") to a nitrocellulose or nylon membrane, and hybridised with a labelled DNA probe complementary to the sequence of interest. By analogy: Northern (RNA), Western (proteins), Eastern (post-translational modifications)

(C) Western blot (for protein)

(D) Eastern blot

Q38. Monoclonal antibodies (mAbs) — crucial diagnostic and therapeutic tools (rituximab, trastuzumab, pembrolizumab) — are produced from:

(A) Pooled human serum

(B) Plant extracts

(C) Hybridomas: fusion (1975, Cesar Milstein and Georges Köhler, Nobel 1984) of a B lymphocyte (which provides antibody specificity but is short-lived) with a myeloma cell (which provides immortality), selected in HAT medium. Each hybridoma clone secretes a single antibody species of defined specificity, indefinitely

(D) Synthetic peptide chemistry

Q39. The conversion of atmospheric N_2 to ammonia in soil ecosystems (biological nitrogen fixation) is carried out by:

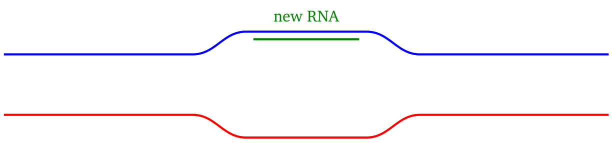
(A) Animals

(B) Fungi alone

(C) Higher plants directly

(D) Specialised prokaryotes: free-living (*Azotobacter*, *Clostridium*, cyanobacteria) and symbiotic (*Rhizobium* in legumes, *Frankia* in actinorhizal plants); all carry Mo-Fe nitrogenase

Q40. During transcription, RNA polymerase opens a localised region of double-stranded DNA to expose the template strand. This open complex, shown below, is called the:



transcription bubble ($\sim$ 17 bp open)

(A) Transcription bubble — $\sim$ 12–17 base pairs of the duplex DNA are melted; the template strand is read by RNA polymerase as it synthesises a complementary RNA strand at $\sim$ 30–50 nt/s in bacteria; behind the bubble, the DNA re-anneals

(B) Replication fork

(C) Holliday junction

(D) Nucleosome

Detailed Solutions

Q1.

Solution

Concept — The tonoplast — plant vacuolar membrane: The large central vacuole (often $> 80\%$ cell volume) is bounded by a single membrane: the tonoplast.

Step 1 — Active transport across tonoplast:

- V-type H^+-ATPase: pumps protons into the lumen using ATP $\rightarrow$ acidic vacuole (pH 4–5.5).
- H^+-pyrophosphatase: a second proton pump, using PP_i (unique to plants).
- Secondary active antiporters use the H^+ gradient to load sugars, Na^+, malate, anthocyanin pigments, defensive metabolites.

Step 2 — Vacuole functions:

- Generates turgor (pushes cytoplasm against cell wall, drives cell expansion, provides mechanical support).
- Storage of ions, sugars, organic acids, pigments (anthocyanin in flowers/fruits).
- Sequestration of toxic secondary metabolites (alkaloids, glucosinolates) released on tissue damage.
- Digestive (lysosome-like) function.
- Waste storage (calcium oxalate crystals).

Final Answer: Tonoplast $\Rightarrow \boxed{C}$

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

Q2.

Solution

Concept — Membrane lipid asymmetry: The two leaflets of the plasma membrane bilayer have systematically different lipid compositions, maintained actively.

Step 1 — The asymmetric distribution:

- **Outer leaflet:** phosphatidylcholine (PC), sphingomyelin (SM), glycolipids.
- **Inner leaflet:** phosphatidylethanolamine (PE), phosphatidylserine (PS, negatively charged), phosphatidylinositol (PI; precursor of signalling phosphoinositides).

- Cholesterol: roughly equal on both sides.

Step 2 — Maintained by three enzyme families:

- Flippases (ATP-driven): move PS, PE from outer to inner leaflet.
- Floppases (ABC transporters): push PC, SM outward.
- Scramblases: Ca^{2+}-activated; randomise distribution during apoptosis or platelet activation.

Step 3 — PS exposure as "eat-me" signal: In apoptotic cells, scramblases expose PS on the outer leaflet; macrophages recognise PS via receptors and engulf the dying cell (efferocytosis). In activated platelets, exposed PS provides the surface for coagulation factor assembly.

Final Answer: PC + SM outside; PE + PS + PI inside $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q2

Q3.

Solution

Concept — Telomerase and the end-replication problem: DNA polymerases work only $5' \to 3'$ and need an RNA primer. The last primer on the lagging strand cannot be replaced by DNA $\Rightarrow$ chromosomes shorten ~ 50–200 bp per cell division.

Step 1 — Telomeres: Tandem repeats of TTAGGG (in vertebrates), ~ 5–15 kb at each chromosome end, coated by shelterin complex (TRF1, TRF2, POT1). Protect ends from being mistaken for DNA breaks.

Step 2 — Telomerase (Greider, Blackburn, Szostak — Nobel 2009): A reverse transcriptase with an integral RNA template:

- TERT: catalytic protein subunit.
- TERC: integral RNA component; template "CCCAAUC" directs TTAGGG addition.
- Extends the $3'$ overhang of chromosome ends; lagging-strand synthesis then fills in.

Step 3 — Expression and disease:

- Active in germ cells, embryonic stem cells, some adult stem cells.
- Silenced in most somatic cells $\to$ Hayflick limit (~ 40–60 divisions).
- Reactivated in ~ 85–90% of cancers (a hallmark).

- Mutations: dyskeratosis congenita, idiopathic pulmonary fibrosis.

Final Answer: Adds TTAGGG repeats using internal RNA template ⇒ $\boxed{B}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept — The spliceosome: A vast and dynamic RNA-protein machine that removes introns and joins exons in pre-mRNA. Discovery of split genes earned Roberts and Sharp the 1993 Nobel.

Step 1 — snRNPs ("snurps"), the building blocks: Five major small nuclear ribonucleoproteins assemble at each intron:

- U1: recognises $5'$ splice site (GU at intron start).
- U2: binds the branch-point adenosine.
- U4/U6/U5 tri-snRNP: catalyses the chemistry.

Step 2 — Two-step transesterification:

(a) $2'$-OH of branch-point A attacks $5'$ splice site $\rightarrow$ lariat intermediate.
(b) $3'$-OH of upstream exon attacks $3'$ splice site $\rightarrow$ exons joined, lariat intron released.

Step 3 — Alternative splicing: $\sim 95\%$ of human multi-exon genes are alternatively spliced. Different exon combinations create multiple protein isoforms from a single gene $\rightarrow$ explains how $\sim 20{,}000$ genes generate $> 100{,}000$ proteins.

Final Answer: Spliceosome (U1, U2, U4/U6/U5 snRNPs) ⇒ $\boxed{B}$

Answer: (B) Go Back to Q4

Q5.

Solution

Concept — Co-translational targeting to the ER: Günter Blobel's signal hypothesis (Nobel 1999): proteins carry intrinsic "address tags" directing them to the correct subcellular destination.

Step 1 — The ER-targeting sequence (events):

(a) Translation begins on a free cytosolic ribosome.

(b) N-terminal signal peptide (15–30 hydrophobic residues) emerges from the ribosome.
(c) Signal Recognition Particle (SRP) binds the signal peptide AND the ribosome; translation arrests temporarily.
(d) Complex docks at the ER membrane via SRP receptor.
(e) Nascent chain feeds through the Sec61 translocon as translation resumes.
(f) Signal peptide cleaved by signal peptidase in the ER lumen.

Step 2 — Other targeting signals (for context):

- NLS: basic-residue clusters; target to nucleus via importin.
- Mitochondrial: N-terminal amphipathic helix; TOM/TIM translocons.
- Peroxisomal: PTS1 (C-terminal SKL) or PTS2.
- Chloroplast: transit peptide.

Step 3 — Diseases of mistargeting: ΔF508 CFTR (cystic fibrosis), I-cell disease, Zellweger syndrome — all reflect defective protein localisation.

Final Answer: N-terminal signal peptide $\rightarrow$ ER via SRP/Sec61 $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q5

Q6.

Solution

Concept — Compartmentalised β-oxidation: Fatty acid β-oxidation occurs in both peroxisomes and mitochondria, with different substrate specificity and chemistry.

Step 1 — Peroxisomal β-oxidation:

- Handles very-long-chain ($\geq C22$) and branched-chain fatty acids (lignoceric, cerotic, phytanic).
- First step: fatty-acyl-CoA oxidase, transferring electrons directly to $O_2 \rightarrow H_2O_2$ (handled by catalase). No ATP captured here.
- Continues until chain shortens to medium length, then exports product to mitochondria (as carnitine ester).

Step 2 — Mitochondrial β-oxidation:

- Handles long-chain ($C12$–$C20$) and medium-chain.
- First step: acyl-CoA dehydrogenase $\rightarrow$ $FADH_2$ feeds ETC $\rightarrow$ ATP.

Step 3 — Peroxisomal disorders:

- X-linked adrenoleukodystrophy: ABCD1 defect; VLCFAs accumulate; neurodegeneration + adrenal failure (the basis of the film *Lorenzo's Oil*).
- Zellweger syndrome: PEX gene defects; peroxisome biogenesis fails; severe and usually fatal in infancy.
- Refsum disease: phytanic acid α-oxidation defect.

Final Answer: Peroxisomes $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q6

Q7.

Solution

Concept — The G_2/M transition driver: MPF: Mitosis-Promoting Factor, discovered by Yoshio Masui (1971) in maturing frog oocytes; biochemistry by Tim Hunt (cyclin) and Paul Nurse (CDK) — Nobel 2001 with Lee Hartwell.

Step 1 — MPF identity: A heterodimer of:

- Cyclin B (regulatory subunit): accumulates through interphase, peaks at mitosis, then destroyed by APC/C-mediated ubiquitination.
- CDK1 (also called Cdc2, catalytic subunit): a serine/threonine kinase, constant throughout the cell cycle.

Step 2 — Activation at G_2/M:

- Cyclin B accumulates in S/G_2.
- Inhibitory phosphorylations on CDK1 keep MPF inactive until G_2/M.
- Cdc25 phosphatase removes inhibitory phosphates $\rightarrow$ MPF active.
- Positive feedback (active MPF activates Cdc25, inhibits Wee1) generates a sharp, switch-like transition.

Step 3 — MPF substrates drive the mitotic state:

- Nuclear lamins (depolymerise $\rightarrow$ nuclear envelope breakdown).
- Histone H1, condensin (chromosome condensation).
- Microtubule-associated proteins (spindle assembly).
- Golgi membrane proteins (Golgi fragmentation).

Step 4 — Inactivation at anaphase: APC/C ubiquitinates cyclin B $\rightarrow$ proteasomal degradation $\rightarrow$ MPF off $\rightarrow$ cell exits mitosis.

Step 5 — Other CDK-cyclin pairs:

- CDK4/6 + cyclin D: G_1 progression.
- CDK2 + cyclin E: G_1/S transition.
- CDK2 + cyclin A: S phase.
- CDK1 + cyclin B: G_2/M (MPF).

Final Answer: MPF (cyclin B + CDK1) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q7

Q8.

Solution

Concept — Cell types of the gastric gland: The stomach mucosa contains specialised secretory cells, each with a distinct product.

Step 1 — Major cell types:

- Mucous neck cells: secrete protective mucus.
- **Parietal (oxyntic) cells:** secrete HCl and intrinsic factor.
- Chief (zymogen) cells: secrete pepsinogen and gastric lipase.
- Enteroendocrine cells (G, ECL, D): secrete hormones (gastrin, histamine, somatostatin).

Step 2 — HCl secretion by parietal cells:

- H^+/K^+-ATPase (the gastric proton pump) on apical canalicular membrane pumps H^+ into lumen in exchange for K^+.
- Cl^- follows passively via apical channels.
- HCO_3^- exits basolaterally in exchange for Cl^- (the "alkaline tide" after a meal).
- Lumen reaches pH $\sim$ 1–2, activating pepsinogen to pepsin and killing pathogens.

Step 3 — Intrinsic factor (IF): A glycoprotein secreted by parietal cells; binds dietary vitamin B_{12}; the IF-B_{12} complex is absorbed in the terminal ileum. Autoimmune destruction of parietal cells $\rightarrow$ *pernicious anaemia* (megaloblastic anaemia + neurological deficits).

Step 4 — Stimulants of acid secretion: Gastrin (from G cells, CCK_B receptors), histamine (from ECL cells, H_2 receptors), and ACh (vagal, M_3 receptors) all converge to activate the proton pump.

Step 5 — Acid-suppression pharmacology:

- Proton pump inhibitors (omeprazole, pantoprazole): irreversibly block H^+/K^+-ATPase; most potent.
- H_2 blockers (ranitidine, famotidine): block H_2 receptors.
- Antacids: neutralise lumen acid.

Final Answer: Parietal (oxyntic) cells $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q8

Q9.

Solution

Concept — Pulmonary surfactant and IRDS: Surfactant replacement therapy is one of the great success stories of modern neonatal medicine.

Step 1 — The physics (Laplace's law): $P = 2T/r$ for a spherical alveolus. Smaller alveoli have higher collapsing pressure (other things being equal). Without surfactant, small alveoli would empty into large ones at end-expiration — alveolar collapse.

Step 2 — Surfactant function:

- Reduces alveolar surface tension (more so at smaller radii, because molecules pack tighter as the alveolus shrinks).
- Prevents collapse (atelectasis).
- Stabilises alveoli of different sizes against each other.
- Raises lung compliance (less work of breathing).

Step 3 — Composition:

- $\sim 90\%$ lipid: chiefly dipalmitoylphosphatidylcholine (DPPC).
- $\sim 10\%$ protein: SP-A, SP-D (hydrophilic, host defence by opsonisation); SP-B, SP-C (hydrophobic, lipid spreading).

Step 4 — Type II pneumocytes: Cuboidal alveolar cells; the surfactant factories; also serve as progenitors of Type I cells after lung injury. Surfactant secretion begins ~ 24 weeks gestation and rises sharply ~ 35 weeks.

Step 5 — IRDS management:

- Antenatal corticosteroids (betamethasone 24–34 weeks): accelerate fetal lung maturation; one of the highest-impact obstetric interventions in history.

- Exogenous surfactant (Survanta, Curosurf, derived from bovine/porcine lung): given intratracheally after birth.
- CPAP or mechanical ventilation as required.

Final Answer: Pulmonary surfactant from Type II pneumocytes ⇒ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept — Cardiac output (CO): The total volume of blood pumped by each ventricle per minute. The fundamental quantitative measure of cardiac performance.

Step 1 — The equation:

$$\boxed{\mathrm{CO} = \mathrm{SV} \times \mathrm{HR}}$$

Step 2 — Resting values (healthy adult):

- HR ~ 70 beats/min.
- SV ~ 70 mL/beat.
- CO ~ 5 L/min (entire blood volume circulates once per minute).
- Right and left ventricle outputs are equal in steady state.

Step 3 — During exercise: CO can rise 5–7 fold in elite athletes (25–35 L/min), via:

- Heart rate increase (up to ~ 200 bpm).
- Stroke volume increase (up to ~ 150 mL): greater venous return $\to$ larger EDV $\to$ Frank-Starling; plus sympathetic-driven contractility increase.

Step 4 — Related parameters:

- Cardiac index: CO normalised to body surface area; resting ~ 3.0 L/min/m^2.
- Ejection fraction (EF) $=$ SV/EDV; normal 55–70%. HFrEF $\leq 40\%$.

Step 5 — Four determinants of stroke volume:

- Preload (EDV; venous return).
- Afterload (aortic pressure to overcome).
- Contractility (intrinsic strength of contraction).

- Heart rate (very high HR shortens diastolic filling, paradoxically lowering SV).

Final Answer: $CO = SV \times HR \Rightarrow \boxed{A}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept — ANP — the heart as an endocrine organ: The discovery by de Bold (1981) that atrial myocytes secrete a hormone overturned the long-held assumption that the heart is just a pump.

Step 1 — Source and stimulus:

- Stored in granules of cardiac atrial myocytes (mainly right atrium).
- Synthesised as preproANP $\rightarrow$ proANP $\rightarrow$ released as 28-aa mature ANP.
- Released in response to atrial stretch (high blood volume, hypertension, heart failure).
- BNP (B-type) is the related ventricular hormone; CNP from endothelium.

Step 2 — Mechanism: ANP binds NPR-A receptor (membrane guanylate cyclase) $\rightarrow$ cGMP $\rightarrow$ effects on kidney, vasculature, adrenal, hypothalamus.

Step 3 — Renal effects (natriuresis + diuresis):

- Vasodilates afferent arteriole, constricts efferent $\rightarrow$ raises GFR.
- Inhibits Na^+ reabsorption in collecting duct (downregulates ENaC).
- Net: increased Na^+ and water excretion.

Step 4 — Other effects:

- Systemic vasodilation $\rightarrow$ lowers BP.
- Inhibits aldosterone (adrenal cortex).
- Inhibits ADH release (hypothalamus).

Net result: lowers blood volume and BP $\rightarrow$ unloads the heart.

Step 5 — Opposes RAAS:

- RAAS: activated when BP/volume low $\rightarrow$ retains Na^+, raises BP.
- ANP/BNP: activated when BP/volume high $\rightarrow$ excretes Na^+, lowers BP.

- Balanced regulatory antagonism.

Step 6 — Clinical use:

- Plasma BNP and NT-proBNP: widely used heart failure biomarkers.
- Sacubitril/valsartan (Entresto): combines an ARB with neprilysin inhibitor (sacubitril) that prolongs natriuretic peptide action; major advance in HFrEF therapy.

Final Answer: Natriuresis, diuresis, lower BP $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q11

Q12.

Solution

Concept — The cerebellum — "little brain": A motor coordinator at the back of the brainstem. Contains ~ 80 billion neurons — more than the rest of the brain combined — yet occupies only $\sim 10\%$ of brain volume.

Step 1 — Anatomy: Two hemispheres + central vermis; cortex with three layers (molecular, Purkinje cell, granular); deep cerebellar nuclei (dentate, interposed, fastigial); three peduncles connecting to brainstem; finely folded surface (folia) for large area.

Step 2 — Functional zones:

- Vestibulocerebellum (flocculonodular lobe): balance, eye movements.
- Spinocerebellum (vermis + paravermis): postural and locomotor coordination.
- Cerebrocerebellum (lateral hemispheres): timing and planning of skilled movements; some cognitive function.

Step 3 — Function as a comparator/predictor: Receives (a) copies of motor commands from motor cortex, (b) sensory feedback from muscles/joints/vestibular/vision. Compares intended with actual movement, computes corrections, predicts forward outcomes. Crucial for motor learning (long-term depression at parallel-fibre $\rightarrow$ Purkinje synapse).

Step 4 — The Purkinje cell: The sole output of cerebellar cortex; massive dendritic tree ($\sim 200{,}000$ parallel-fibre inputs); GABAergic (inhibitory); signals to deep cerebellar nuclei $\rightarrow$ thalamus $\rightarrow$ motor cortex.

Step 5 — Lesion syndrome:

- Ataxia (uncoordinated movement).
- Intention tremor (oscillation on reaching).
- Dysmetria (over/undershoot).
- Dysdiadochokinesia (impaired rapid alternating movement).
- Nystagmus.
- Lesions cause IPSILATERAL signs.
- Causes: stroke, MS, alcoholic cerebellar degeneration, hereditary ataxias (SCA), tumours.

Step 6 — Beyond motor: The "cognitive cerebellum": contributes to language, working memory, attention, emotion (Schmahmann syndrome with cerebellar lesions).

Final Answer: Cerebellum $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q12

Q13.

Solution

Concept — Aldosterone, principal mineralocorticoid: A steroid hormone secreted by the zona glomerulosa of the adrenal cortex; the endpoint of the RAAS axis.

Step 1 — Triggers for secretion:

- Angiotensin II (the major stimulus, downstream of low renal perfusion).
- Hyperkalaemia (high plasma K^+) — direct stimulus.
- ACTH (minor, transient).

Step 2 — Mechanism: Lipophilic steroid; crosses cell membranes; binds intracellular mineralocorticoid receptor (MR) in principal cells of DCT and cortical collecting duct $\rightarrow$ MR enters nucleus $\rightarrow$ transcribes target genes:

- Apical: more ENaC (Na^+ channels), more ROMK (K^+ channels).
- Basolateral: more Na^+/K^+-ATPase.
- Net: Na^+ reabsorbed (water follows), K^+ excreted.

Step 3 — Effects:

- Increased blood Na^+ and water retention $\rightarrow$ raised blood volume and BP.
- Increased K^+ excretion.
- Increased H^+ excretion.

Step 4 — Disorders:

- Primary hyperaldosteronism (Conn syndrome): adrenal adenoma; hypertension + hypokalaemia.
- Secondary hyperaldosteronism: from RAAS activation (renal artery stenosis, heart failure, cirrhosis).
- Addison's disease: low aldosterone (and cortisol) $\rightarrow$ hypotension, hyperkalaemia, salt craving.

Step 5 — Pharmacology: Spironolactone, eplerenone: MR antagonists; used in heart failure, primary aldosteronism, resistant hypertension. ACE inhibitors and ARBs indirectly lower aldosterone.

Final Answer: Na^+ reabsorption, K^+ excretion $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q13

Q14.

Solution

Concept — Leaf abscission: The orderly shedding of leaves at the right time, mediated by a specialised abscission zone.

Step 1 — The abscission zone (AZ): A narrow band of small parenchymatous cells at the petiole base. Divided into a separation layer (where cells separate) and a protective layer (which later seals the leaf scar with suberin and lignin).

Step 2 — Hormonal control:

- **Auxin retards abscission:** an actively photosynthesising young leaf produces IAA that suppresses the AZ.
- **Ethylene promotes abscission:** when auxin declines (leaf ageing, stress), the AZ becomes sensitive to ethylene; ethylene induces cellulases and polygalacturonases in the AZ $\rightarrow$ middle lamella dissolves $\rightarrow$ leaf separates.
- ABA may sensitise the AZ via promoting senescence.

Step 3 — Autumn leaf fall in deciduous trees:

- Shortening day length triggers senescence.
- Chlorophyll degraded; nutrients (N, P) translocated back to stem.
- Accessory pigments revealed $\rightarrow$ yellow/orange/red colours.
- Auxin levels drop, ethylene rises $\rightarrow$ AZ activates, leaves drop, scar sealed.

Step 4 — Adaptive value: Reduces winter water loss when soil water is frozen; conserves nutrients; lowers metabolic cost during dormancy.

Step 5 — Commercial: Ethephon used to encourage uniform fruit drop in mechanical harvesting (cherries, olives). Anti-ethylene treatments (1-MCP, silver thiosulfate) extend cut-flower shelf life.

Final Answer: Ethylene promotes; auxin retards ⇒ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept — Recessive epistasis: the Bombay phenotype: Epistasis = one gene masks the expression of another at a different locus.

Step 1 — ABO biochemistry:

- All ABO antigens are built on a precursor: the **H antigen** (a fucose-terminated oligosaccharide on RBCs).
- The H gene (FUT1, chromosome 19) encodes fucosyltransferase, which builds the H antigen.
- The ABO gene (chromosome 9) then modifies H:
  - I^A: adds N-acetylgalactosamine → A antigen.
  - I^B: adds galactose → B antigen.
  - i: no addition (H remains → blood type O).

Step 2 — Bombay phenotype:

- Genotype hh at H locus → no fucosyltransferase → no H antigen.
- Without H precursor, A and B antigens cannot be built, regardless of ABO genotype. An I^AI^A hh person has full ABO machinery but no substrate to act on.
- Such people TYPE as "O" but they actually have a unique phenotype.

Step 3 — Crucial clinical implication: Bombay individuals carry anti-A, anti-B AND anti-H antibodies in their serum (the anti-H is the critical difference). They CANNOT receive ordinary group-O blood (which expresses H antigen) — their anti-H would react. They can only receive blood from another Bombay donor. Hence Bombay donors are kept on special registries.

Step 4 — Discovery: Y. M. Bhende and colleagues at K. E. M. Hospital, Bombay (now Mumbai), 1952, found a few individuals whose serum reacted with all standard O blood — the new phenotype was named after the city.

Step 5 — Compare with other gene interactions:

- Pleiotropy: ONE gene $\rightarrow$ many traits.
- Polygenic inheritance: MANY genes $\rightarrow$ one continuous trait.
- Epistasis: one gene's allele masks another gene's expression.
- Linkage: two genes physically close, inherited together.

Final Answer: Recessive epistasis (hh masks ABO) $\Rightarrow \boxed{D}$

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

Q16.

Solution

Concept — Crick's central dogma (1958, formalised 1970): The fundamental rule governing how genetic information flows between the three major informational biopolymers.

Step 1 — The classical pathway:

$$\boxed{\text{DNA} \xrightarrow{\text{transcription}} \text{RNA} \xrightarrow{\text{translation}} \text{Protein}}$$

- DNA stores genetic information.
- Transcribed to RNA by RNA polymerase.
- Translated to protein by ribosomes using the genetic code (codon $\rightarrow$ amino acid).

Step 2 — The forbidden flows:

- Protein $\rightarrow$ DNA: never observed.
- Protein $\rightarrow$ RNA: never observed.
- Protein $\rightarrow$ Protein (without nucleic acid intermediate): prions are a partial exception (see below).

Step 3 — Exceptions discovered later:

- **Reverse transcription** (RNA $\rightarrow$ DNA): retroviruses (HIV, HTLV) use reverse transcriptase. Discovered by Temin and Baltimore (1970, Nobel 1975).

- **RNA replication** (RNA $\rightarrow$ RNA): RNA viruses (polio, influenza, SARS-CoV-2) use RNA-dependent RNA polymerases.
- **Prion propagation:** misfolded prion proteins recruit normal cellular PrP to misfold — a protein-only "inheritance" of conformation, but no sequence change.
- **Epigenetic inheritance:** methylation patterns transmitted at cell division; DNA sequence unchanged.

Final Answer: DNA $\rightarrow$ RNA $\rightarrow$ Protein $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q16

Q17.

Solution

Concept — The ribosome A/P/E sites: The ribosome (~ 2.5 MDa, $\sim 65\%$ RNA), determined by Yonath/Ramakrishnan/Steitz (Nobel 2009), has three tRNA binding sites at the subunit interface.

Step 1 — The three sites:

- **A (Aminoacyl) site:** where incoming aminoacyl-tRNAs enter, paired with the mRNA codon.
- **P (Peptidyl) site:** holds the tRNA bearing the growing peptide chain.
- **E (Exit) site:** holds the deacylated tRNA before it departs.

Step 2 — Elongation cycle (one amino acid added):

(a) **Decoding:** aminoacyl-tRNA, escorted by EF-Tu·GTP, enters A site. If anticodon matches codon, GTP hydrolysed, EF-Tu released.

(b) **Peptidyl transfer:** the A-site amino acid's α-amino group attacks the P-site peptidyl carbonyl carbon $\rightarrow$ peptide bond. Polypeptide now extends from A-site tRNA.

(c) **Translocation:** EF-G·GTP drives ribosome forward by one codon. A $\rightarrow$ P, P $\rightarrow$ E, E exits.

Step 3 — Peptidyl transfer is ribozymal: Catalysed by 23 S rRNA in the large subunit — no protein essential. The ribosome is a **ribozyme**. Strong evidence for the RNA-world hypothesis.

Step 4 — Antibiotics targeting ribosome:

- Tetracyclines: block A-site aminoacyl-tRNA binding.

- Aminoglycosides (streptomycin, gentamicin): cause A-site misreading.
- Chloramphenicol: blocks peptidyl transfer.
- Macrolides (erythromycin, azithromycin): block translocation/exit tunnel.
- Linezolid: blocks 50 S subunit assembly.

Final Answer: A (aminoacyl) site $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q17

Q18.

Solution

Concept — Genetic drift, bottleneck and founder effect: Random changes in allele frequencies in finite populations. Sewall Wright (1931) developed the theory.

Step 1 — Bottleneck vs founder effect: Both are special cases of genetic drift.

- **Bottleneck effect:** a once-large population is collapsed to a small number by catastrophe (disease, climate, hunting); when it expands again, allele frequencies of the rebuilt population differ from the original. Cheetahs: severe bottleneck $\sim 12{,}000$ years ago; remarkably low genetic variation today.
- **Founder effect:** a small group breaks off from a parent population to colonise a new area. By chance, the founders carry an unrepresentative sample of the parent gene pool; the new population's allele frequencies differ.

Step 2 — Human examples of founder effect:

- Ellis-van Creveld syndrome high frequency among the Pennsylvania (Lancaster) Amish, descended from ~ 200 founders.
- Huntington's disease at Lake Maracaibo, Venezuela; traced to a single 19th-century woman.
- Porphyria variegata high prevalence among South African Afrikaners; traced to a single 17th-century Dutch immigrant couple.
- Tay-Sachs disease in Ashkenazi Jews.
- Familial hypercholesterolaemia in French Canadians.

Step 3 — Mathematical sense: Genetic drift is stronger in smaller populations. The variance in allele frequency change per generation is $p(1-p)/2N_e$, where N_e is the effective population size. A founder group of 10 has $50\times$ the drift per generation as a population of 500.

Step 4 — Implications:

- Founder effect can cause recessive disease alleles to reach unusually high frequency in isolated populations.
- Useful for population genetics — isolated communities are valuable for gene-disease mapping (e.g. Finland, Iceland).
- Distinct from natural selection (which acts on phenotype) — both can change allele frequencies but founder/drift are random.

Final Answer: Founder effect ⇒ C

Answer: (C) Go Back to Q18

Q19.

Solution

Concept — The Biological Species Concept (BSC): Formalised by Ernst Mayr (1942, in *Systematics and the Origin of Species*); the dominant species concept in modern evolutionary biology.

Step 1 — Definition: A species = a group of **actually or potentially interbreeding** natural populations **reproductively isolated** from other such groups.

Step 2 — The operational criterion: Reproductive isolation. Two populations are different species if they cannot produce fertile, viable offspring together under natural conditions.

Step 3 — Reproductive isolating mechanisms:

- **Prezygotic (prevent fertilisation):**
  - Ecological (different habitats).
  - Temporal (different breeding seasons).
  - Behavioural (different courtship signals).
  - Mechanical (incompatible reproductive structures).
  - Gametic (sperm and egg incompatibility).
- **Postzygotic (prevent fertile offspring):**
  - Hybrid inviability (zygote dies early).
  - Hybrid sterility (e.g. mule from horse × donkey).
  - Hybrid breakdown (F_2 generation weak/sterile).

Step 4 — Limitations of BSC:

- Not applicable to asexual organisms (bacteria, archaea, many protists).
- Cannot be applied directly to fossils.

- Cannot be applied to allopatric populations (geographically isolated; we don't know whether they would interbreed).
- Hybridisation between "good species" is common in plants (and in some animal groups).

Step 5 — Alternative species concepts:

- Morphological (typological): based on observable form.
- Phylogenetic / cladistic: smallest monophyletic group.
- Ecological: occupies a distinct niche.
- Genetic: distinct gene pool by sequence data.

Final Answer: Interbreeding natural populations, reproductively isolated $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q19

Q20.

Solution

Concept — SRY — the master switch of mammalian male development: The Y chromosome's most famous gene. Discovered 1990 by Peter Goodfellow and Robin Lovell-Badge.

Step 1 — Bipotential gonad: Until ~ 7 weeks of human gestation, the gonad is bipotential — could become either testis or ovary. The default differentiation is to ovary (no SRY needed).

Step 2 — SRY action:

- SRY (Sex-determining Region Y) is a single small gene on the short arm of the Y chromosome.
- Encodes a transcription factor (Testis-Determining Factor, TDF) with an HMG-box DNA-binding domain.
- Brief expression at ~ 7 weeks gestation in the indifferent gonad's somatic cells.
- Activates downstream genes (notably SOX9) that drive Sertoli-cell differentiation $\rightarrow$ seminiferous-cord formation $\rightarrow$ testis development.
- Without SRY (XX): no testis $\rightarrow$ default ovary development.

Step 3 — Hormonal sequence in male development:

- Sertoli cells secrete anti-Müllerian hormone (AMH/MIS) $\rightarrow$ regression of Müllerian ducts (which would form female internal genitalia).

- Leydig cells produce testosterone → Wolffian ducts develop into epididymis, vas deferens, seminal vesicles.
- Testosterone converted to dihydrotestosterone (DHT, by 5α-reductase) in target tissues → external male genitalia.

Step 4 — Disorders:

- Swyer syndrome (46,XY gonadal dysgenesis): SRY mutated → phenotypic female with streak gonads.
- XX male syndrome: SRY translocated to an X chromosome during paternal meiosis → phenotypic male.
- Androgen insensitivity syndrome: 46,XY with mutant androgen receptor → female external phenotype.

Final Answer: SRY (Sex-determining Region Y) ⇒ $\boxed{A}$

Answer: (A) Go Back to Q20

Q21.

Solution

Concept — Cleavage and the morula stage: Cleavage = rapid mitotic divisions of the zygote without intervening growth; cell number rises while total volume stays constant; individual cells (blastomeres) become smaller.

Step 1 — Cleavage timeline in humans:

- Day 0: fertilisation (zygote, 1 cell).
- Day 1: first cleavage → 2 cells.
- Day 2: 4 cells.
- Day 3: 8 cells; compaction begins (E-cadherin-mediated cell-cell contacts maximise).
- Day 4: **morula** ($\sim$ 16 cells, solid mulberry-like ball, still within the zona pellucida). Latin *morus* = mulberry.
- Day 5: cavitation begins → blastocyst (inner cell mass + trophoblast + fluid-filled blastocoel).
- Day 6–7: blastocyst hatches from zona; implants.

Step 2 — Compaction and cell polarisation: At ~ 8 cells, blastomeres maximise contact, gain apical-basal polarity, and begin to differ. Outer cells (which retain outer surface) → trophoblast; inner cells → inner cell mass (ICM, the future embryo).

Step 3 — Why morula is distinct:

- Solid ball (no cavity yet), unlike blastula.
- Still within zona pellucida (no implantation yet).
- Cells starting to differentiate (compaction) but not yet committed.

Step 4 — Clinical relevance:

- In IVF, transfer can occur at cleavage stage (day 3, ~ 8 cells) or blastocyst stage (day 5). Blastocyst-stage transfer has higher implantation rates.
- Pre-implantation genetic testing typically biopsies cells at day 5 from the trophoblast.
- Twinning: monozygotic twins arise from a single zygote that separates at 1-cell, 2-cell, morula, or blastocyst stages, with different patterns of membrane sharing depending on when separation occurs.

Final Answer: Morula $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q21

Q22.

Solution

Concept — Gastrulation — forming the germ layers: The morphogenetic events of the 3rd week of human development; transforms a bilaminar disc into a trilaminar disc with three primary germ layers.

Step 1 — The primitive streak: A linear midline thickening of the epiblast appears at $\sim$ day 15. Cells of the epiblast migrate to the streak, ingress through it, and emerge as the underlying germ layers.

Step 2 — Three germ layers established:

- **Ectoderm:** remains on top; later gives rise to epidermis, nervous system (neural tube), neural crest derivatives.
- **Mesoderm:** the middle layer; gives rise to muscles, bones, dermis, kidneys, gonads, heart, vasculature, blood.
- **Endoderm:** ingresses deepest; lines the gut tube and gives rise to GI epithelium, liver, pancreas, lungs (epithelium), thyroid, urinary bladder.

Step 3 — Body-axis establishment:

- Anterior-posterior axis: established by node and primitive streak.

- Dorsal-ventral axis: BMP/chordin antagonism.
- Left-right asymmetry: cilia in the node create a leftward flow; signalling cascades break the L-R symmetry.

Step 4 — Why gastrulation is so important:

- Lewis Wolpert: "It is not birth, marriage, or death, but gastrulation which is truly the most important time in your life".
- Sets up the basic body plan from which all organ systems develop.
- Errors lead to severe congenital malformations (caudal regression syndrome, situs inversus, conjoined twinning).

Step 5 — Next stage — neurulation: The ectoderm overlying the notochord (mesodermal axial rod) folds into the neural tube (the future brain and spinal cord). Failure causes neural tube defects (anencephaly, spina bifida). Folate supplementation in pregnancy dramatically reduces NTD incidence.

Final Answer: Establishes three germ layers (ectoderm, mesoderm, endoderm) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q22

Q23.

Solution

Concept — Placental antibody transfer and immunoglobulin classes: Five immunoglobulin classes in humans, each with distinct function and structural features.

Step 1 — The five classes:

- **IgG** (monomer, $\sim 75\%$ of serum Ig): main antibody of secondary immune response; **the only class transported across the placenta**; long half-life (~ 21 days); four subclasses (IgG1, 2, 3, 4).
- **IgM** (pentamer): first antibody made in primary response; high avidity due to 10 binding sites; activates complement strongly; too large to cross placenta.
- **IgA** (dimer in secretions, monomer in serum): mucosal immunity; secreted in saliva, tears, breast milk, gut; protects infant's gut as "passive immunity" via breast milk.
- **IgE** (monomer): mediates type-I allergy; binds high-affinity FcϵRI on mast cells; very low serum concentration.

- **IgD** (monomer): mostly a B-cell receptor; serum function unclear.

Step 2 — Placental IgG transfer:

- Active transport by neonatal Fc receptor (FcRn) on syncytiotrophoblast.
- Begins around week 14, accelerates through third trimester.
- Newborn IgG levels match (sometimes exceed) maternal levels.
- Provides ~ 6 months of protection while infant's own immune system matures.
- Mediates resistance to many infections (measles, mumps, varicella, tetanus, etc.) that the mother had or was vaccinated against.

Step 3 — Postnatal — IgA in breast milk: Mother's secretory IgA (sIgA) coats the infant's gut mucosa, providing passive immunity at the most common entry point for pathogens. Breastfeeding has well-documented infection-protective effects.

Step 4 — Clinical relevance:

- Maternal IgG can cause haemolytic disease of the newborn (Rh incompatibility): an Rh-negative mother previously sensitised to Rh-positive fetal blood develops anti-D IgG that crosses placenta and destroys foetal RBCs in subsequent pregnancies. Prevented by anti-D Rh immunoglobulin (RhoGAM) prophylaxis.
- Maternal autoimmune antibodies (anti-Ro/SSA, anti-La/SSB, anti-platelet) can cause neonatal lupus, congenital heart block, or neonatal thrombocytopenia.
- IVIG (intravenous immunoglobulin, mostly IgG) used to treat many immune disorders.

Final Answer: IgG $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q23

Q24.

Solution

Concept — Medical Termination of Pregnancy (MTP) Act, India: Originally enacted in 1971 in India; amended in 2021 with significant expansion. Among the more liberal abortion laws globally, balancing women's rights with public health.

Step 1 — Original 1971 MTP Act:

- Up to 12 weeks: opinion of one Registered Medical Practitioner (RMP) required.
- 12–20 weeks: opinion of two RMPs required.
- Permitted indications: risk to mother's life or health (physical/mental); foetal abnormality; failure of contraception (married women only); pregnancy due to rape.

Step 2 — 2021 amendment (current law):

- Up to 20 weeks: opinion of one RMP suffices.
- 20–24 weeks: opinion of two RMPs, for special categories: rape survivors, incest, minors, mentally ill women, women with physical disabilities, women whose marital status changes during pregnancy (widowed/divorced), foetal abnormalities, humanitarian/emergency situations.
- Beyond 24 weeks: only with recommendation of a Medical Board for substantial foetal abnormalities. Each State maintains such Boards.
- Confidentiality of the woman's identity made stricter.
- Contraceptive failure: now applies to ANY woman or her partner (not only married women).

Step 3 — Methods of MTP:

- Early ($\leq$ 9 weeks): medical abortion — mifepristone (antiprogestin) followed by misoprostol (prostaglandin analogue); very safe and effective.
- Up to 12 weeks: vacuum aspiration (MVA / EVA).
- 13–20 weeks: dilation and evacuation (D&E).
- Late: medical induction (prostaglandins).

Step 4 — Public health importance:

- Unsafe abortion remains a major cause of maternal mortality globally.
- Liberalisation of abortion law + access to safe services dramatically reduces maternal deaths.

- Sex-selective abortion remains a serious problem in India; the PCPNDT Act (1994) prohibits pre-conception/pre-natal sex determination for the purpose of sex selection.

Final Answer: Up to 20 weeks (one RMP), 20–24 weeks (two RMPs, special categories), > 24 for foetal abnormalities via Medical Board $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q24

Q25.

Solution

Concept — Anther wall layers and microsporogenesis: A typical mature dicot/monocot anther has four wall layers around each pollen sac (microsporangium), each with distinct function.

Step 1 — Layers from outside to inside:

(a) **Epidermis:** outermost, single layer; protective; eventually stretches and tears at dehiscence.

(b) **Endothecium:** below epidermis; cells develop characteristic fibrous (lignified) thickenings on their inner walls. As the anther dries, these thickenings cause asymmetric contraction $\rightarrow$ anther splits open along the stomium $\rightarrow$ pollen released. Without functional endothecium, the anther cannot dehisce.

(c) **Middle layers (1–3):** ephemeral; usually crushed and resorbed as pollen matures; contribute to nutrition of developing microspores.

(d) **Tapetum:** innermost; directly in contact with microspore mother cells. Provides nutrition, secretes callase to release microspores from callose tetrads, and supplies sporopollenin (the chemically resistant biopolymer of the pollen exine). Tapetum is short-lived: degenerates after pollen maturation.

Step 2 — Tapetum types:

- Secretory (parietal) tapetum: cells remain in place; secrete material into the locule. Most common.
- Amoeboid (periplasmodial) tapetum: cell walls break down; cytoplasm extends among the microspores.

Step 3 — Tapetum significance: Defective tapetum $\rightarrow$ male sterility. Cytoplasmic male sterility (CMS) results from defective mitochondria in tapetal cells; exploited in commercial F_1 hybrid seed production (maize, rice, sunflower).

Step 4 — Microsporogenesis (microspore formation): Microspore mother cells (microsporocytes, $2n$) undergo meiosis $\to$ four haploid microspores in a tetrad surrounded by callose; callase from the tapetum releases individual microspores. Each microspore undergoes a single asymmetric mitotic division to form a pollen grain with a larger vegetative cell and a smaller generative cell (which later divides to form the two sperm).

Final Answer: Epidermis $\to$ endothecium $\to$ middle layers $\to$ tapetum $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q25

Q26.

Solution

Concept — Megasporogenesis — formation of the megaspores: Female counterpart of microsporogenesis. Takes place inside the ovule, deep within the ovary.

Step 1 — The ovule: A complex structure attached to the placenta of the ovary by a stalk (funicle):

- Nucellus: the central mass of tissue; contains the megasporocyte.
- Integuments (1 in monocots/most angiosperms, 2 in many others): surrounding protective layers.
- Micropyle: a small opening at the apex (where pollen tube enters).
- Chalaza: opposite the micropyle.

Step 2 — Megasporogenesis steps:

(a) A single hypodermal cell of the nucellus differentiates into the megaspore mother cell (megasporocyte, $2n$).
(b) Megasporocyte undergoes meiosis $\to$ four haploid megaspores arranged in a linear tetrad.
(c) Three megaspores typically degenerate; only one (usually the chalazal one) survives as the **functional megaspore**.

Step 3 — Megagametogenesis (the embryo sac): The functional megaspore undergoes three successive mitotic divisions WITHOUT cytokinesis $\to$ 8 haploid nuclei in a coenocyte $\to$ then cellularises into the classic seven-celled, eight-nucleate Polygonum-type embryo sac:

- Three antipodal cells at the chalazal end.
- Two central polar nuclei (later fuse to form a $2n$ secondary nucleus, important for endosperm).

- Two synergid cells flanking the egg (guide pollen tube; secrete chemoattractants).
- One **egg cell** at the micropylar end (the female gamete).

Step 4 — Variation: Other types of embryo-sac development exist (Allium-, Adoxa-, Drusa-, Fritillaria-types) differing in which megaspores survive and how many nuclei come from which megaspore. The Polygonum type is most common ($\sim 70\%$ of angiosperms).

Step 5 — Double fertilisation: At fertilisation, one sperm fuses with the egg ($\rightarrow 2n$ zygote $\rightarrow$ embryo); the second sperm fuses with the two polar nuclei ($\rightarrow 3n$ primary endosperm nucleus $\rightarrow$ endosperm). Unique to angiosperms.

Final Answer: Megasporocyte meiosis $\rightarrow$ four megaspores; one functional, three degenerate $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q26

Q27.

Solution

Concept — Ethylene's role in seed germination: While ABA enforces dormancy, ethylene (the only gaseous plant hormone) breaks dormancy in many species — especially under anoxic conditions.

Step 1 — Examples where ethylene promotes germination:

- Rice (especially submerged seeds): ethylene accumulates in the water-saturated submerged seed; activates expression of submergence-tolerance genes; promotes coleoptile elongation through the water column.
- Lettuce: dark-imbibed seeds; ethylene can substitute for the red-light requirement.
- Sunflower: ethylene breaks dormancy.
- Cereal grains (in some species): ethylene induces α-amylase activity in aleurone.

Step 2 — Mechanism:

- Ethylene receptors (ETR1-family) are negative regulators of signalling; binding ethylene relieves their inhibition.
- Downstream: EIN2, EIN3/EIL transcription factors activated.
- Cross-talk with ABA: ethylene counteracts ABA-induced dormancy.
- In rice: SUB1A gene (submergence tolerance) is ethylene-responsive.

Step 3 — Practical applications:

- Ethephon (a chemical that releases ethylene when applied to plants): used to synchronise germination of stored seeds.
- Storage with ethylene-absorbent material: extends seed dormancy.
- In wild plants, ethylene from microbial decomposition of nearby dead biomass may signal favourable post-fire/disturbance conditions for germination.

Step 4 — Other hormones in seed dormancy/germination:

- ABA: enforces dormancy.
- GA: opposes ABA, promotes germination (induces α-amylase in barley aleurone $\rightarrow$ basis of malting).
- Cytokinin: promotes germination in some species.
- Ethylene: see above.

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

Answer: (D) Go Back to Q27

Q28.

Solution

Concept — Phototropism — shoot bending toward light: The classical case study in plant hormone biology. The original demonstration involves Charles Darwin and his son Francis (1880, in *The Power of Movement in Plants*).

Step 1 — Darwin's experiment (1880):

- Grass coleoptiles (the protective sheath around the emerging shoot) bent toward unilateral light.
- Covering the tip with an opaque cap prevented bending; covering the base did not.
- Conclusion: the light-perceiving region (the tip) and the bending region (just below) are different; a signal must travel from tip to bending region.

Step 2 — Boysen-Jensen, Paal, Went experiments: A series of agar-block experiments by Boysen-Jensen (1913), Paal (1919), and Frits Went (1928) showed:

- A diffusible chemical signal from the tip is responsible.
- This chemical was named "auxin" (Greek *auxein* = to grow) and later identified as indole-3-acetic acid (IAA).

Step 3 — The Cholodny-Went hypothesis (1926):

- Light is perceived by phototropin (a blue-light flavoprotein receptor) at the tip.
- PIN auxin-efflux carriers redistribute laterally: more on the shaded side, less on the lit side.
- Auxin accumulates on the shaded side.
- Auxin promotes cell elongation $\rightarrow$ shaded side grows faster than lit side $\rightarrow$ shoot bends toward light.

Step 4 — The acid-growth hypothesis (cell elongation):

- Auxin activates plasma membrane H^+-ATPases.
- Proton extrusion acidifies the cell wall.
- Acid-activated expansins loosen wall polymers.
- Turgor pressure drives water uptake and cell expansion.

Step 5 — Receptors:

- Phototropins (phot1, phot2): blue/UV-A light receptors; LOV domains autophosphorylate on light absorption $\rightarrow$ kinase cascade.
- Cryptochromes (cry1, cry2): also blue-light receptors; involved in photomorphogenesis, circadian clock, but less in phototropism.
- Phytochromes: red/far-red receptors.

Final Answer: Auxin (IAA) accumulates on shaded side $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q28

Q29.

Solution

Concept — Rhodophyta (red algae): Predominantly marine algae, especially abundant in tropical and subtropical waters. Phycoerythrin's red colour absorbs blue/green light, which penetrates deeper into water than other wavelengths — allowing red algae to photosynthesise at greater depths than green algae.

Step 1 — Photosynthetic pigments:

- Chlorophyll a (universal).
- Phycoerythrin (red protein-pigment) and phycocyanin (blue) — biliprotein accessory pigments organised into phycobilisomes (also in cyanobacteria).

The red colour is dominant.

- No chlorophyll b (unlike green algae).

Step 2 — Other diagnostic features:

- Cell wall: cellulose plus polysaccharides (agar in *Gracilaria*, *Gelidium*; carrageenan in *Chondrus*; alginic acid in some).
- Stored food: floridean starch (an amylopectin-like glucan, stored in cytoplasm not plastid).
- No flagellated stages at any point in the life cycle (unique among algae).
- Often complex life cycles with three alternating phases (triphasic alternation: gametophyte, carposporophyte, tetrasporophyte).

Step 3 — Habitat and depth: Some red algae (*Phycoseris*, certain corallines) grow at 200 m depth, the deepest of any photosynthetic organisms, exploiting the blue-green light that penetrates clear water. Many are calcified (deposit $CaCO_3$); coralline red algae are major reef-builders alongside corals.

Step 4 — Economic uses:

- **Nori**: dried *Pyropia/Porphyra* sheets, used to wrap sushi; major Japanese aquaculture industry.
- **Agar**: from *Gelidium* and *Gracilaria*; thickener, microbiology medium (agarose).
- **Carrageenan**: from *Chondrus crispus* (Irish moss); food thickener (dairy products), pharmaceuticals.
- Dietary supplements rich in iodine, minerals.
- Recently: aquaculture of red algae is being expanded for biofuel, bioremediation, and carbon sequestration.

Step 5 — Evolutionary significance: Red algae diverged early from the green-plant lineage. Their plastids resulted from primary endosymbiosis of a cyanobacterium. They gave rise (via secondary endosymbiosis) to the plastids of cryptophytes, haptophytes, diatoms, brown algae — a major branch of eukaryotic photosynthesis.

Final Answer: Chlorophyll a + phycoerythrin + phycocyanin; no flagella; floridean starch $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q29

Q30.

Solution

Concept — Phylum Annelida — segmented worms: $\sim$ 22,000 species. Latin *annulus* = small ring; segmental rings are the visible feature.

Step 1 — Diagnostic features:

- Bilaterally symmetric, triploblastic, true coelomate (eucoelomate).
- **Metameric segmentation:** body composed of a longitudinal series of similar segments (metameres) separated internally by septa. Each segment has its own pair of nephridia, ganglia, gonads (in some), and locomotor muscles.
- Body wall: cuticle (collagen) + epidermis + circular and longitudinal muscle layers.
- Closed circulatory system (most), with dorsal and ventral longitudinal vessels and lateral connectives.
- Excretory: nephridia (per segment).
- Nervous: ventral nerve cord with segmental ganglia; cerebral ganglia ("brain") anteriorly.
- Setae (chaetae): chitinous bristles; aid in locomotion (esp. in Oligochaeta).

Step 2 — Three traditional classes:

- **Polychaeta** ("many setae"): marine; well-developed parapodia (lateral fleshy lobes for swimming); separate sexes; examples: *Nereis* (clam worm), *Aphrodita* (sea mouse), tube worms.
- **Oligochaeta** ("few setae"): mostly terrestrial/freshwater; few setae; hermaphrodite; cocoon-forming; examples: earthworms (*Lumbricus terrestris*, *Pheretima posthuma*).
- **Hirudinea** (leeches): no setae (most); anterior and posterior suckers; ectoparasites or predators; hermaphrodite; *Hirudo medicinalis* (used medicinally for its hirudin anticoagulant).

Step 3 — Ecological importance:

- Earthworms are crucial soil engineers — aerate soil, mix organic matter (Darwin wrote his last book about them), enrich agricultural soils (vermicomposting).
- Marine polychaetes: dominant in benthic and pelagic communities; food source for fish and seabirds.

Step 4 — Compare with other "worm" phyla:

- Platyhelminthes (flatworms): acoelomate, unsegmented.
- Nematoda (roundworms): pseudocoelomate, unsegmented.
- Annelida: eucoelomate, segmented.

Final Answer: True coelom + metameric segmentation $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q30

Q31.

Solution

Concept — Class Aves (birds): $\sim$ 10,000 described living species. Highly successful vertebrate class with worldwide distribution; descendants of theropod dinosaurs.

Step 1 — Diagnostic synapomorphies:

- **Feathers**: keratin protein; modified epidermal scales; unique to Aves among living animals. Functions: flight, insulation, display, waterproofing, brood patches.
- Forelimbs modified into **wings** (in flightless species, reduced or modified).
- Hollow **pneumatic bones**: laced with air sacs; light yet strong; allow flight.
- Endothermic (warm-blooded); high metabolic rate.
- Four-chambered heart: complete separation of pulmonary and systemic circuits.
- Oviparous: lay calcified hard-shelled amniotic eggs.
- **Unique respiratory system:** parabronchial lungs with through-flow ventilation via air sacs; much more efficient O_2 extraction than mammalian tidal lungs.
- Beak (rhamphotheca) instead of teeth (most groups); no jawbones with teeth in living birds.
- Single ovary (left) in most females (the right one is reduced) — weight saving for flight.

Step 2 — Reproductive biology:

- Internal fertilisation.
- Eggs laid externally; incubated by parent's body heat (brood patches).
- Bi-parental care common.
- Long parent-offspring association; altricial vs precocial chicks.

Step 3 — Evolutionary origin:

- Living birds belong to Theropoda (the same dinosaur clade as *T. rex*); they are technically "avian dinosaurs".
- *Archaeopteryx* (~ 150 Mya, late Jurassic): the iconic transitional fossil with teeth, clawed wings, long bony tail, AND feathers.
- Recent discoveries (China): many feathered non-avian theropods (*Microraptor*, *Sinosauropteryx*).

Step 4 — Major orders: Passeriformes (perching birds, $\sim 60\%$ of bird species), Accipitriformes (eagles, hawks), Strigiformes (owls), Galliformes (chickens, turkeys), Anseriformes (ducks, geese), Sphenisciformes (penguins), Struthioniformes (ostriches).

Final Answer: Feathers + wings + hollow pneumatic bones + four-chambered heart + parabronchial lungs $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q31

Q32.

Solution

Concept — Cholera and cholera toxin: A waterborne diarrhoeal disease that has caused seven pandemics since the early 19th century. The seventh, ongoing since 1961, includes the 2010 Haiti outbreak.

Step 1 — Pathogen: *Vibrio cholerae*: a curved (comma-shaped), motile, gram-negative bacillus; aerobic to facultative anaerobic; survives in brackish water and marine environments. Pathogenic strains belong to serogroups O1 (biotypes Classical and El Tor) and O139.

Step 2 — Transmission: Faecal-oral via contaminated water and food. Massive doses required (10^8 bacteria) because gastric acid kills most ingested vibrios; outbreaks linked to poor sanitation, contaminated water supplies, post-disaster settings.

Step 3 — Pathogenesis — the cholera toxin (CT) story:

- Vibrios reach the small intestine, adhere via toxin-coregulated pilus (TCP), produce cholera toxin.
- CT is an AB_5 toxin: 5 B subunits bind GM1 ganglioside on enterocyte surface; A subunit enters the cell.
- Inside the cell, the A subunit ADP-ribosylates the $G\alpha_s$ subunit of the adenylate cyclase regulatory G protein $\rightarrow$ locks it in the "on" state $\rightarrow$ persistently high cAMP.

- High cAMP → phosphorylates and activates CFTR Cl^- channel → massive Cl^- efflux into the lumen; Na^+, water and other electrolytes follow.
- Net: torrential, isotonic, "rice-water" diarrhoea, up to 20 L/day — catastrophic dehydration, hypotension, hypovolaemic shock, death within hours if untreated.

Step 4 — Clinical features:

- Sudden profuse watery diarrhoea (no blood, no fever typically).
- Severe vomiting.
- Rapid dehydration: sunken eyes, decreased skin turgor, weak pulse, hypotension.
- Death can occur within 4–12 hours of severe disease.

Step 5 — Treatment and prevention:

- **Oral rehydration solution (ORS):** a simple glucose-electrolyte solution; revolutionised diarrhoeal disease management; uses the SGLT1 Na^+/glucose co-transporter (still functional during cholera) to drive Na^+ and water absorption against the toxin-driven secretion. Saved millions of lives. "Perhaps the most important medical advance this century" — *The Lancet*, 1978.
- IV fluids in severe cases.
- Antibiotics (doxycycline, azithromycin) shorten symptoms.
- Oral cholera vaccines (Dukoral, Shanchol, Euvichol).
- Sanitation and safe water supplies — the long-term solution.

Step 6 — Historical note: John Snow's 1854 Broad Street pump investigation in London established cholera as waterborne — foundational moment in epidemiology and public health. Vibrio cholerae was isolated by Robert Koch (1883).

Final Answer: *Vibrio cholerae* ⇒ $\boxed{A}$

Answer: (A) Go Back to Q32

Q33.

Solution

Concept — The complement system: A collection of ~ 30 plasma and cell-surface proteins forming a cascade that amplifies and integrates innate and adaptive immunity. Discovered by Jules Bordet (1895, Nobel 1919); named for "complementing" antibody action.

Step 1 — Three activation pathways converge:

- **Classical pathway:** triggered by C1q binding to IgG/IgM bound to antigen on pathogen surface. Antibody-dependent.
- **Lectin pathway:** triggered by mannose-binding lectin (MBL) recognising sugar patterns (mannose, fucose) on microbial surfaces. Antibody-independent.
- **Alternative pathway:** spontaneous, low-level C3 hydrolysis amplified on pathogen surfaces that lack regulatory proteins. Antibody-independent.

Step 2 — Common pathway and effector functions: All three converge at C3 cleavage to C3a + C3b. Then:

- **Opsonisation:** C3b binds pathogen surface; phagocytes (with complement receptor CR1) recognise and engulf opsonised pathogens.
- **Anaphylatoxins:** C3a and C5a recruit and activate inflammatory cells (mast cells, neutrophils); cause vasodilation, capillary permeability.
- **Membrane Attack Complex (MAC):** C5b + C6 + C7 + C8 + multiple C9 assemble a transmembrane pore in the pathogen membrane $\rightarrow$ osmotic lysis. Particularly effective against gram-negative bacteria (notably *Neisseria*).

Step 3 — Regulation: Host cells protect themselves with regulators: DAF (decay-accelerating factor), CD59 (MAC inhibitor), MCP (membrane cofactor protein), factor H, factor I. Defects lead to:

- Paroxysmal nocturnal haemoglobinuria (PIGA mutation $\rightarrow$ defective GPI-anchored DAF/CD59 $\rightarrow$ complement-mediated RBC lysis).
- Atypical haemolytic uraemic syndrome (factor H deficiency).

Step 4 — Complement deficiencies and disease:

- C1, C4, C2 deficiency: SLE-like syndrome (immune complex clearance impaired).
- C3 deficiency: severe recurrent pyogenic infections.

- Terminal (C5-C9) deficiency: recurrent *Neisseria* infections (meningococcal disease).
- C1 inhibitor deficiency: hereditary angioedema.

Step 5 — Therapeutic targeting: Eculizumab (anti-C5 monoclonal antibody) blocks MAC formation; used for paroxysmal nocturnal haemoglobinuria, aHUS, and some neurological autoimmune diseases.

Final Answer: Membrane Attack Complex (MAC) $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q33

Q34.

Solution

Concept — Hepatic ethanol catabolism: Two cytosolic and mitochondrial enzymes are responsible for the bulk of alcohol metabolism in humans. The pathway provides both energy and a substantial source of NADH.

Step 1 — The two-step pathway:

$$\text{Ethanol} \xrightarrow[\text{NAD}^+ \to \text{NADH}]{\text{Alcohol dehydrogenase (ADH)}} \text{Acetaldehyde} \xrightarrow[\text{NAD}^+ \to \text{NADH}]{\text{Aldehyde dehydrogenase (ALDH2)}} \text{Acetate}$$

Step 2 — ADH (alcohol dehydrogenase):

- Cytosolic zinc-containing enzyme; uses NAD^+.
- Multiple isoforms in humans (ADH1, 4, 5, 7).
- Converts ethanol $\rightarrow$ acetaldehyde (a toxic intermediate).
- Saturated at relatively low ethanol concentrations $\rightarrow$ zero-order kinetics (constant rate of $\sim 100\,\text{mg/kg/h}$ in adults).

Step 3 — ALDH2 (mitochondrial aldehyde dehydrogenase):

- Mitochondrial NAD^+-dependent enzyme.
- Converts acetaldehyde $\rightarrow$ acetate (which enters general metabolism as acetyl-CoA).
- Low Km for acetaldehyde; normally keeps acetaldehyde levels very low.

Step 4 — The "Asian flush" (alcohol flush reaction): About 40–50% of East Asians carry the ALDH2$*$2 variant (Glu487Lys); the resulting enzyme has only $\sim 8\%$ of normal activity. Heterozygotes after drinking accumulate acetaldehyde

→ facial flushing, tachycardia, headache, nausea. The same trait substantially increases risk of oesophageal cancer with heavy drinking. Mendelian randomisation studies use ALDH2$*$2 to estimate causal effects of alcohol on disease risk.

Step 5 — The alternative MEOS pathway: The microsomal ethanol oxidising system (MEOS; CYP2E1) becomes important at high ethanol levels and after chronic use. CYP2E1 is inducible → increased tolerance with chronic drinking; but also increased free radical generation, contributing to alcoholic liver damage.

Step 6 — Disulfiram (Antabuse): A drug used in alcoholism treatment; inhibits ALDH2 → acetaldehyde accumulates after alcohol → unpleasant reaction; aversive conditioning.

Step 7 — NADH overload: Chronic alcohol overproduces NADH → inhibits gluconeogenesis (fasting hypoglycaemia), promotes lactic acidosis, lipogenesis (fatty liver). Underlies much of alcoholic liver disease pathology.

Final Answer: ADH → acetaldehyde → ALDH2 → acetate ⇒ $\boxed{C}$

Answer: (C) Go Back to Q34

Q35.

Solution

Concept — Fundamental vs realised niche: A central concept in community ecology. Joseph Grinnell (1917) introduced "niche"; Charles Elton (1927) refined it; G. Evelyn Hutchinson (1957) formalised the n-dimensional hypervolume concept of niche.

Step 1 — Definitions:

- **Fundamental niche:** the full range of physical (temperature, light, humidity) and biological (food availability) conditions under which a species could persist in the absence of competitors and predators. The theoretical maximum.
- **Realised niche:** the subset of the fundamental niche actually occupied by the species in the wild, constrained by interspecific competition, predation, parasitism and disease.

Step 2 — Connell's barnacle experiment (1961) — the classical demonstration: On rocky Scottish coast intertidal zones:

- *Chthamalus stellatus* (smaller barnacle): **fundamental niche** extends across the entire intertidal range it could tolerate physiologically.

- *Balanus balanoides* (larger, faster-growing barnacle): occupies the lower-mid intertidal.
- Where they overlap, Balanus competitively excludes Chthamalus (smothers and undercuts it).
- Result: Chthamalus's **realised niche** is limited to the upper intertidal where Balanus cannot tolerate desiccation. Removing Balanus experimentally expanded Chthamalus into the lower zone.

Step 3 — Competitive exclusion principle (Gause, 1934): "Two species with identical niches cannot coexist indefinitely; the better competitor will exclude the other". Coexistence requires niche differentiation (e.g. different food, different microhabitats, different activity periods).

Step 4 — Niche partitioning examples:

- Robert MacArthur's warbler study: 5 warbler species coexist in spruce trees in New England, each foraging at different heights/zones.
- Tropical reef fishes: different feeding strategies (herbivores, planktivores, piscivores).
- Crepuscular vs diurnal vs nocturnal mammals partition the same habitat by time.

Step 5 — Implications for invasive species: An invasive species in a new range may exploit a wider niche (closer to its fundamental) because competitors/predators that constrained it in its native range are absent — explaining why invasive populations often grow explosively.

Final Answer: The actually occupied subset of the fundamental niche $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q35

Q36.

Solution

Concept — Food webs and trophic complexity: Real ecosystems are not linear food chains but interconnected food webs with many cross-links. The web view, pioneered by Charles Elton, is foundational to ecosystem ecology.

Step 1 — Linear food chain vs food web:

- Food chain: simple linear sequence (e.g. grass $\to$ grasshopper $\to$ frog $\to$ snake $\to$ hawk). A simplification.
- Food web: many interconnected food chains; species feed at multiple trophic

levels and on multiple species; multiple species can feed on the same prey.

Step 2 — Why webs better describe reality:

- Most consumers are omnivorous: bears eat berries (primary consumer) and salmon (secondary). Humans eat plants and animals.
- A single species is typically eaten by multiple predators.
- Decomposers and detritivores feed on dead matter from all levels.
- Many parasites span multiple hosts.

Step 3 — Implications:

- **Stability:** food webs are more resilient than linear chains. If one species is removed, alternative pathways absorb the loss. However, highly connected webs can also propagate disturbance widely.
- **Keystone species:** disproportionately important relative to their abundance. Removing them causes cascading effects. Robert Paine's classic experiment: removing the sea star *Pisaster ochraceus* from rocky intertidal $\rightarrow$ mussel monoculture takes over $\rightarrow$ biodiversity crashes.
- **Trophic cascades:** top-down effects of predators through multiple levels. Wolves in Yellowstone (re-introduced 1995): reduced elk numbers $\rightarrow$ allowed willows and aspens to regenerate $\rightarrow$ beavers returned $\rightarrow$ riparian zones recovered.
- **Biomagnification:** fat-soluble persistent pollutants (DDT, mercury, PCBs) accumulate up the food web, reaching highest concentrations in top predators (eagles, ospreys, tuna, polar bears).

Step 4 — Network analysis: Modern ecology uses graph theory to analyse food webs — characterising connectance, modularity, nestedness, robustness, etc. Some metrics (e.g. "trophic level" of a species, defined as $1+$ weighted average trophic level of its prey) require web analysis, not just chain logic.

Final Answer: Most consumers feed on multiple species at multiple trophic levels $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q36

Q37.

Solution

Concept — The Southern blot and its derivatives: A landmark molecular biology technique invented by Edwin Southern at the University of Edinburgh (1975). Allows specific DNA fragments among many to be identified by hybridisation.

Step 1 — Southern blot procedure:

(a) Digest genomic (or other) DNA with one or more restriction enzymes.
(b) Separate the fragments by agarose gel electrophoresis (by size).
(c) Denature the DNA in the gel (alkali; separates strands).
(d) Transfer ("blot") the single-stranded fragments from the gel to a nitrocellulose or nylon membrane by capillary action — preserving the fragment pattern.
(e) Hybridise the membrane with a labelled (radioactive or fluorescent) DNA probe complementary to the sequence of interest.
(f) Wash off non-specific binding; detect the probe by autoradiography or fluorescence imaging.

Step 2 — "Blotting" family (named by analogy, geographic-direction joke):

- **Southern blot:** DNA; the original (named after Edwin Southern).
- **Northern blot:** RNA; detects specific mRNAs.
- **Western blot:** protein; detects specific proteins using antibodies (e.g. HIV confirmatory test).
- **Eastern blot:** protein post-translational modifications (less standardised).
- **Southwestern blot:** DNA-protein interactions.

Step 3 — Applications of Southern blot:

- Detecting gene rearrangements, deletions, amplifications.
- Restriction fragment length polymorphism (RFLP) analysis: pre-PCR DNA fingerprinting; was the basis of paternity testing and early forensic DNA analysis.
- Genomic-library screening.
- Verifying transgene insertion in transgenic animals/plants.
- Detecting Huntington's disease (CAG repeat expansion).

Step 4 — Modern alternatives:

- PCR-based methods are faster and require less DNA.

- qPCR for quantitative detection.
- Next-generation sequencing has largely supplanted Southern blot for many applications.
- However, Southern blot remains useful for verifying transgene structure (e.g. in plant biotech regulatory dossiers) and detecting large structural variations.

Final Answer: Southern blot $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q37

Q38.

Solution

Concept — Monoclonal antibodies and the hybridoma: Cesar Milstein and Georges Köhler (1975, Nobel Prize 1984) developed the hybridoma technique — one of the most consequential innovations in 20th-century biology, opening therapy, diagnostics, and basic research.

Step 1 — The problem to solve: In a normal immune response, the body produces a polyclonal mixture of many different antibodies recognising different epitopes of an antigen. Each B-cell clone makes one specific antibody. But primary B cells are short-lived in culture (a few days), so you cannot maintain a single antibody-producing clone indefinitely.

Step 2 — The hybridoma solution:

(a) Immunise a mouse with the antigen of interest; activated B-cell clones develop in spleen.
(b) Harvest spleen cells (containing the desired B cells).
(c) Fuse spleen cells with a myeloma cell line (immortal but cannot make functional antibody and is HGPRT-deficient) using polyethylene glycol (PEG) or electrofusion.
(d) Plate the fused cells in HAT medium (hypoxanthine + aminopterin + thymidine):
    - Aminopterin blocks the de novo nucleotide pathway.
    - Cells survive only if they use the salvage pathway (which requires HGPRT).
    - Unfused myeloma cells (HGPRT-deficient) die in HAT.
    - Unfused B cells die naturally in a few days.
    - Only fused cells (hybridomas: immortal + HGPRT-positive) survive.
(e) Screen hybridoma colonies for antibody specificity (ELISA).

(f) Pick a single positive clone → hybridoma line that secretes ONE specific antibody (monoclonal) forever.

Step 3 — Therapeutic monoclonal antibodies: A multi-billion-dollar industry. Major examples:

- **Rituximab** (anti-CD20): B-cell lymphomas, autoimmune diseases.
- **Trastuzumab** (Herceptin, anti-HER2): HER2-positive breast cancer.
- **Bevacizumab** (anti-VEGF): cancers, age-related macular degeneration.
- **Adalimumab** (anti-TNFα): rheumatoid arthritis, IBD, psoriasis.
- **Pembrolizumab, nivolumab** (anti-PD-1): immune checkpoint inhibitors revolutionising cancer immunotherapy.
- **Anti-COVID neutralising antibodies**: pandemic-era therapy.

Step 4 — Humanisation: Mouse antibodies cause anti-mouse responses in humans. Engineering:

- Chimeric: $\sim 65\%$ human Fc + mouse Fab (e.g. rituximab; suffix -ximab).
- Humanised: only mouse CDR loops grafted onto human framework (e.g. trastuzumab; suffix -zumab).
- Fully human: from transgenic mice with human Ig genes, or by phage display (e.g. adalimumab; suffix -umab).

Step 5 — Diagnostic applications:

- Pregnancy tests (anti-hCG).
- Blood typing (anti-A, anti-B).
- Flow cytometry markers (CD3, CD4, CD8, etc.).
- Histopathology immunostaining.
- ELISA, lateral flow tests.

Final Answer: Hybridoma (fusion of B cell + myeloma) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q38

Q39.

Solution

Concept — Biological nitrogen fixation: A reaction exclusive to certain prokaryotes; one of the most ecologically and economically important biochemical processes on Earth.

Step 1 — The reaction: Conversion of dinitrogen gas (N_2, very stable due to the triple bond) into a biologically usable form (ammonia / ammonium):

$$N_2 + 8\,H^+ + 8\,e^- + 16\,ATP \rightarrow 2\,NH_3 + H_2 + 16\,ADP + 16\,P_i$$

Catalysed by the nitrogenase enzyme complex (a Mo-Fe + Fe-protein system); extremely energy-expensive (16 ATP per N_2); irreversibly inactivated by oxygen.

Step 2 — Why only prokaryotes: The nitrogenase enzyme system has not been found in any eukaryote (animals, plants, fungi, protists). All biological nitrogen fixation depends on prokaryotes; eukaryotes that benefit (e.g. legumes) do so through symbiosis.

Step 3 — Diazotrophs (N_2-fixers):

- **Free-living aerobic:** *Azotobacter*, *Beijerinckia* (soil).
- **Free-living anaerobic:** *Clostridium* (soil).
- **Cyanobacteria:** *Nostoc*, *Anabaena*, *Calothrix* (in heterocysts).
- **Symbiotic with legumes:** *Rhizobium*, *Bradyrhizobium*, *Sinorhizobium* (in root nodules; provides 20–50 g N/m^2/yr in well-nodulated fields).
- **Symbiotic with actinorhizal plants:** *Frankia* (in nodules of *Alnus*, *Casuarina*).
- **Symbiotic with cycads:** cyanobacteria in coralloid roots.
- **Symbiotic with *Azolla* water fern:** *Anabaena azollae* (used as green manure in rice paddies).

Step 4 — Industrial fixation — Haber-Bosch process (1909/1913): The chemical synthesis of NH_3 from N_2 and H_2 at high T ($\sim 450\,°C$) and P (~ 200 atm) with Fe catalyst. Produces ~ 175 million tonnes of N annually (more than biological fixation!) — feeds $\sim 50\%$ of the human population through synthetic fertiliser. Energy-intensive: ~ 1–2% of global energy consumption.

Step 5 — Environmental issues: Excess agricultural N causes eutrophication (algal blooms, "dead zones" in Gulf of Mexico, Baltic Sea), nitrate contamination of groundwater, and nitrous oxide emissions (a potent greenhouse gas, $\sim 300\times$ CO_2).

Step 6 — Engineering N_2-fixing crops? Active research goal: engineer cereals to fix nitrogen, either by introducing nitrogenase, by inducing root-nodule symbiosis (as in legumes), or by association with endophytic diazotrophs. Would massively reduce global fertiliser need.

Final Answer: Specialised prokaryotes (free-living and symbiotic) $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q39

Q40.

Solution

Concept — The transcription bubble: The locally unwound region of DNA where RNA polymerase reads the template strand to synthesise mRNA.

Step 1 — Structure:

- $\sim$ 12–17 base pairs of the DNA duplex are melted (unwound) at the active site of RNA polymerase.
- One strand (the **template strand**, also called antisense strand) is read $3' \to 5'$; the other strand (the coding/sense strand) is displaced.
- RNA is synthesised $5' \to 3'$ complementary to the template.
- Behind the bubble (upstream of polymerase), the DNA re-anneals.
- In front of the bubble (downstream), the DNA is being progressively unwound.

Step 2 — The RNA-DNA hybrid: Inside the bubble, the nascent RNA forms a transient $\sim$ 8–10 bp RNA-DNA hybrid with the template, before peeling off and emerging from the polymerase.

Step 3 — Transcription kinetics:

- Bacterial RNA pol: $\sim$ 30–50 nt/s.
- Eukaryotic Pol II: somewhat slower ($\sim$ 10–30 nt/s), with frequent pausing for regulation.
- Polymerase has intrinsic $3'$–$5'$ exonuclease (proofreading) and backtracking capability.

Step 4 — Topological problem: Unwinding $\sim$ 17 bp introduces positive supercoils ahead and negative supercoils behind the polymerase (the "twin-supercoiled-domain" model). Topoisomerases relax these supercoils:

- Topo I (or DNA gyrase reverse activity): relaxes negative supercoils behind.

- Topo II / DNA gyrase: relaxes positive supercoils ahead.

Antibiotics targeting bacterial DNA gyrase: fluoroquinolones (ciprofloxacin, levofloxacin); cancer drugs targeting topo II: etoposide, doxorubicin.

Step 5 — Transcription factors and the bubble: In eukaryotes, the bubble at promoters forms through the action of general transcription factor TFIIH, which contains DNA helicase activity (XPB, XPD subunits). Mutations in XPB/XPD cause xeroderma pigmentosum, Cockayne syndrome.

Final Answer: Transcription bubble $\Rightarrow \boxed{A}$

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

Answer Key

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