tr joelPCM Academic Council

DEPARTMENT OF NATURAL SCIENCES · ADVANCED BIOLOGY

Advanced Biology — Paper 1: Theory | Set II

MODEL ANSWERS & WORKED SOLUTIONS · MARK SCHEME

For Examiner / Teacher Use Only | Total: 80 marks | BIO/P1/SET-II/MS/2025

MARK SCHEME NOTES

1. Each bullet point (✓) represents 1 mark unless otherwise stated.
2. Accept any equivalent correct biological reasoning, even if differently phrased.
3. Credit annotated diagrams that convey the same information as a written point.
4. Contextual application to the Ugandan scenario earns credit alongside biological knowledge.
5. Do not penalise for additional correct information beyond the mark scheme.

SECTION A — COMPULSORY

Items 1 & 2 | 40 marks

ITEM 1 — Model Answer

AO1 · Molecular Biology, DNA Technology & Cell Division

AO1 **20 marks**

Part (a) — Molecular mechanisms of ULV disruption (12 marks)

12 marks available

How the retrovirus integrates and hijacks cell machinery:

- ✓ ULV is a retrovirus carrying a single-stranded RNA genome. After entry into host cells, viral reverse transcriptase synthesises a complementary DNA (cDNA) strand from the RNA template, then creates a double-stranded DNA (dsDNA) provirus — this is the mechanistic basis for reverse transcriptase activity detected in Groups B and C.
- ✓ Integrase enzyme inserts the proviral dsDNA into the host cell's chromosomal DNA at random integration sites. Group B shows 1–3 integration sites; Group C shows 8–15, reflecting viral replication cycles over time.
- ✓ The proviral DNA is transcribed by the host's RNA polymerase II, producing viral mRNAs translated by host ribosomes into viral proteins — this is why viral RNA copies increase from 120–400 (Group B) to 4,200–8,600 (Group C).

Disruption of tumour-suppressor gene expression:

- ✓ Integration near the tumour-suppressor gene disrupts its regulatory region (promoter or enhancer sequences) by physical interruption or by inserting viral promoters/enhancers that interfere with transcription factor binding — hence tumour-suppressor mRNA falls to 64% (Group B) and 11% (Group C).
- ✓ Reduced mRNA leads directly to reduced tumour-suppressor protein (58% in Group B; 7% in Group C), as less template is available for ribosomal translation at the rough ER.
- ✓ The tumour-suppressor protein normally phosphorylates and activates downstream effectors that hold the cell at the G1 checkpoint (spindle assembly checkpoint proteins, CDK inhibitors

such as p21). With only 7% of normal protein in Group C, G1 checkpoint arrest falls from 82% to 12%.

Loss of cell cycle control and uncontrolled division:

- ✓ Passage through G1 unchecked allows cells with DNA damage and viral insertions to enter S phase — the cell commits to DNA replication and subsequent mitosis regardless of genomic integrity.
- ✓ Mitotic index rises from 0.04 (Group A) → 0.12 (Group B) → 0.61 (Group C), reflecting an exponentially growing proportion of cells undergoing uncontrolled division — consistent with oncogenic transformation.
- ✓ Multiple integration events (Group C: 8–15 sites) increase the probability of disrupting additional tumour suppressor or proto-oncogene loci in subsequent replication cycles, further removing brakes on cell division.
- ✓ PCR detects ULV at <50 copies/mL in both Groups B and C — demonstrating that PCR's amplification of specific viral sequences (via denaturation, primer annealing, and Taq polymerase extension cycles) is sensitive enough to detect infection long before clinical signs appear.

Award max 12. Require: reverse transcription mechanism (2 marks), integration and gene disruption (3 marks), mRNA/protein link (2 marks), cell cycle loss (3 marks), data use (2 marks). Accept diagram of retroviral cycle for up to 3 marks.

Part (b) — UBTS screening strategy (8 marks)

8 marks available

- ✓ Implement mandatory PCR-based screening of all donated blood using ULV-specific primers targeting the reverse transcriptase gene — PCR's sensitivity (<50 copies/mL) ensures detection in early-stage donors (Group B) before protein levels change, preventing contaminated blood entering the supply.
- ✓ Use real-time quantitative PCR (qPCR) with a fluorescent probe (e.g., TaqMan) to quantify viral load — allows discrimination between low-level early infection and high-load advanced infection, enabling clinical risk stratification of donors.
- ✓ Introduce a confirmed nucleic acid testing (NAT) window: even PCR has a brief pre-detection window. Retain donor samples for 14 days and re-test if initial result is borderline — prevents false-negative releases.
- ✓ Establish a national donor deferral registry: permanently defer any donor confirmed ULV-positive. Link UBTS centres across Uganda through a shared database to prevent re-donation at a different centre.
- ✓ Screen recipient blood banks for proviral DNA using nested PCR on stored samples dating back 12 months before ULV was identified — perform look-back investigation to identify possibly infected transfusion recipients and offer them monitoring and treatment.
- ✓ Develop a serological ELISA assay for ULV surface antigens as a low-cost, high-throughput complementary screen — ELISA detects antibody–antigen binding, allowing rapid preliminary screening of large sample batches with PCR confirmation of positives.
- ✓ Train blood bank staff at all regional transfusion centres in biosafety protocols for handling ULV-positive samples — viral samples represent a contamination risk; standard precautions (PPE, Class II biosafety cabinets) must be enforced.
- ✓ Engage the Uganda Virus Research Institute (UVRI) to sequence isolates from positive donors — whole-genome sequencing of ULV strains informs primer design updates if the virus mutates, ensuring PCR assays remain effective as the virus evolves.

Award max 8. Require: PCR mechanism with justification (2), at minimum two further distinct strategies (4), and a population-level safety measure (2). Do not award marks for strategies without biological justification.

ITEM 1 TOTAL: 20 marks

ITEM 2 — Model Answer

AO2 · Plant Physiology, Photosynthesis & Agricultural Adaptation

AO2 **20 marks**

Part (a) — Differences in photosynthesis, chlorophyll, stomata and yield (12 marks)

12 marks available

Light-dependent reactions and chlorophyll ratios:

- ✓ In the light-dependent stage, photons excite electrons in chlorophyll molecules in the thylakoid membranes. In deep shade (5%), very few photons reach the leaf — photosystem II and PSI are rarely activated; hence net photosynthesis is only 0.8 µmol $CO_2/m^2/s$.
- ✓ Under deep shade, plants express more chlorophyll b (low chl a:b ratio of 1.8:1) — chlorophyll b absorbs shorter wavelengths (450 nm, blue) efficiently and transfers energy to reaction centres, maximising photon capture in dim light. Full sun plants have chl a:b of 3.8:1, indicating proportionally more chlorophyll a for direct photochemistry at high irradiance.
- ✓ At 30% shade (moderate), light intensity matches cocoa's natural understorey optimum — net photosynthesis rises to 6.4 µmol $CO_2/m^2/s$; the 60% shade treatment achieves the peak of 9.1 µmol $CO_2/m^2/s$, near cocoa's light saturation point.
- ✓ In full sun (100%), photon flux exceeds the light saturation point (950 µmol/m^2/s). Excess excitation energy cannot be used in the electron transport chain — reactive oxygen species (ROS) accumulate, causing photoinhibition of PSII. Net photosynthesis drops to 7.3 µmol $CO_2/m^2/s$ despite maximum light, explaining lower yield than 60% shade.

Light-independent reactions (Calvin cycle) and CO_2 supply:

- ✓ The Calvin cycle in the stroma uses ATP and NADPH from the light reactions to fix CO_2 via RuBisCO into glycerate-3-phosphate (G3P) and ultimately sucrose/starch. In deep shade, low ATP/NADPH limits G3P reduction — this is the biochemical basis for the low compensation and saturation points in shade-grown plants.
- ✓ Stomatal conductance peaks at 60% shade (0.31 mol/m^2/s), maximising CO_2 entry for the Calvin cycle while transpiration remains manageable. Under full sun, stomata close partially (conductance drops to 0.18) due to heat and ABA-mediated water stress — restricting CO_2 supply and further suppressing the Calvin cycle.

Stomatal behaviour and water balance:

- ✓ Full sun raises leaf temperature and vapour pressure deficit dramatically — guard cells lose turgor (leaf water potential −1.6 MPa vs −0.3 MPa in deep shade), triggering ABA synthesis that causes K^+ efflux from guard cells, closing stomata. This explains leaf scorch incidence of 38% under full sun.
- ✓ Deep shade plants maintain near-zero water stress (−0.3 MPa) but cannot generate sufficient ATP for active growth despite open stomata — hence lowest yield (180 kg/ha).

Yield explanation:

- ✓ Maximum yield at 60% shade (890 kg/ha) reflects the optimal balance: sufficient light for high photosynthesis, open stomata for CO_2 entry, and no photoinhibition or heat stress. Full-sun yield (640 kg/ha) is lower because photoinhibition and stomatal closure limit carbon assimilation and divert resources to stress repair rather than bean development.

> *Award max 12. Require: chlorophyll b/shade adaptation (2), light reactions mechanism (2), Calvin cycle CO_2 link (2), stomatal physiology/ABA (2), photoinhibition explanation (2), yield synthesis from data (2). Credit chl a/b diagram if correctly labelled.*

Part (b) — Agroforestry strategy for Bundibugyo farmers (8 marks)

8 marks available

- ✓ Adopt 60% shade agroforestry — interplant cocoa with native shade trees (e.g., Maesopsis eminii, Albizia species) spaced to achieve approximately 60% canopy cover. Data show this maximises yield (890 kg/ha) by providing optimal photon flux without photoinhibition, and suppresses leaf scorch from 38% to 9%.
- ✓ Select nitrogen-fixing shade trees (Albizia, Acacia) — through Rhizobium root nodule symbiosis these trees fix atmospheric N2 into ammonium ions, enriching soil nitrogen availability for cocoa's amino acid and chlorophyll synthesis, reducing need for costly synthetic fertilisers.
- ✓ Implement shade tree pruning schedules timed to match dry season — during dry spells, increase shade cover to reduce leaf water potential stress on cocoa (prevent reaching −1.6 MPa); during cooler, humid periods, thin canopy to allow closer to 100% sun to boost photosynthesis rate temporarily.
- ✓ Apply organic mulch under cocoa — decomposing leaf litter improves soil water-holding capacity, moderating soil moisture fluctuations. This maintains stomatal conductance above 0.19 mol/m²/s even during dry periods, preventing Calvin cycle limitation.
- ✓ Introduce banana or plantain as medium-height shade layer in a multi-strata system — provides additional 20–30% shade to prevent full-sun conditions, with the added benefit of a food/income crop for farmers, increasing farm system resilience.
- ✓ Establish cocoa nurseries using 60%-shade propagation — ensures seedlings develop high chlorophyll b content and efficient shade-adapted light harvesting from the outset, reducing transplant shock when placed under forest canopy.
- ✓ Train farmers to monitor leaf water potential indicators (leaf rolling, afternoon wilting) as simple proxies for stomatal stress — allows timely supplemental irrigation at critical bean-fill stages to prevent yield collapse during dry spells.
- ✓ Advocate for carbon credit payments to farmers who maintain shade-tree agroforestry — trees sequester carbon (above-ground biomass), qualifying farms for REDD+ or voluntary carbon market payments, providing an additional economic incentive over full-sun monoculture.

> *Award max 8. Require: justification with reference to data or physiological mechanism for each strategy. Minimum 4 distinct strategies for full marks. Do not accept strategies without biological or agronomic justification.*

ITEM 2 TOTAL: 20 marks

ITEM 3 — Model Answer

AO3 · Gas Transport, Respiratory Physiology & Environmental Adaptation

AO3 **20 marks**

Part (a) — Mechanisms of oxygen delivery at altitude (12 marks)

12 marks available

Effect of reduced atmospheric pO2 at altitude:

- ✓ At 5,109 m, atmospheric pressure is approximately 54% of sea level. Partial pressure of oxygen (pO2) falls proportionally — arterial pO2 of non-acclimatised climbers drops to 42 mmHg vs 95 mmHg at 1,400 m. On the oxyhaemoglobin dissociation curve, 42 mmHg falls on the steep portion, meaning haemoglobin saturation drops dramatically — tissue oxygen delivery is severely compromised.
- ✓ Non-acclimatised climbers show acute hyperventilation (28 breaths/min vs 14 at sea level) — peripheral chemoreceptors (carotid bodies) detect falling pO2 and signal the medullary respiratory centre to increase ventilation rate, partially compensating by raising alveolar pO2.
- ✓ Hyperventilation causes CO2 washout → respiratory alkalosis (blood pH rises) → left-shift of the oxyhaemoglobin dissociation curve → haemoglobin loads O2 more readily in the lungs, partially offsetting the low pO2. However, this also impairs O2 unloading at tissues.

Bakonzo highlanders — chronic physiological adaptations:

- ✓ Chronic hypoxia stimulates hypoxia-inducible factor (HIF-1α) → upregulates erythropoietin (EPO) gene expression in the kidneys. Data confirm: Bakonzo EPO level is 34 mIU/mL vs 8 in lowlanders — driving erythropoiesis in bone marrow, raising haemoglobin to 18.6 g/dL and haematocrit to 58%.
- ✓ Higher haematocrit increases the oxygen-carrying capacity of blood — more haemoglobin molecules per unit volume means more O2 transported per litre of blood, compensating for low arterial pO2.
- ✓ Bakonzo haemoglobin has a higher O2 affinity (P50 = 22 mmHg vs 26 mmHg in lowlanders) — the dissociation curve is left-shifted. At the low alveolar pO2 of high altitude (approx. 50 mmHg), haemoglobin loads significantly more O2 than normal-affinity haemoglobin at the same pO2.
- ✓ Low 2,3-BPG (13.1 µmol/g Hb) in Bakonzo — 2,3-BPG normally binds deoxy-Hb and right-shifts the curve. Lower 2,3-BPG in Bakonzo favours O2 loading in the lungs. This contrasts with non-acclimatised climbers (22.8 µmol/g Hb), where acutely elevated 2,3-BPG right-shifts the curve to aid O2 unloading at tissues — a short-term acute response.

Bar-headed goose — extreme high-altitude adaptation:

- ✓ P50 of only 14 mmHg in bar-headed goose haemoglobin — exceptionally high O2 affinity due to a single amino acid substitution (Pro → Ala at α119) in the haemoglobin α-chain, weakening 2,3-BPG binding and increasing O2 affinity. This allows the goose to load haemoglobin at alveolar pO2 as low as 30 mmHg.
- ✓ The goose also has large parabronchial lungs with a cross-current gas exchange mechanism — unlike mammalian tidal ventilation, avian cross-current exchange maintains a higher effective pO2 gradient from air capillaries to blood throughout the entire cycle, maximising O2 extraction even at extreme altitudes.
- ✓ Ventilation rate of 36 breaths/min at 7,000 m maintains alveolar pO2 above the haemoglobin's very high-affinity loading threshold — even tiny amounts of O2 in the air are captured efficiently.

Award max 12. Require: dissociation curve mechanics with P50 (3 marks), 2,3-BPG mechanisms (2 marks), EPO/haematocrit pathway (2 marks), bar-headed goose molecular basis (2 marks), ventilation chemoreceptor link (2 marks), data synthesis (1 mark).

Part (b) — Adaptive significance and clinical management (8 marks)

8 marks available

- ✓ Evolutionary adaptive significance: Bakonzo highland populations have been under directional selection for thousands of years — individuals with naturally higher EPO responsiveness, higher haemoglobin affinity alleles, and greater vasodilation capacity survived and reproduced better at altitude. The current phenotype represents accumulated polygenic adaptations to chronic hypoxia.
- ✓ The bar-headed goose adaptation represents more extreme evolutionary selection over millions of years of high-altitude migration — a single structural gene mutation in α-globin conferred such a strong fitness advantage (ability to migrate over the Himalayas rather than around them, saving energy) that it became fixed in the population.
- ✓ Acute Mountain Sickness (AMS) management — prescribe acetazolamide prophylactically before ascent: this carbonic anhydrase inhibitor induces metabolic acidosis (mild), stimulating peripheral chemoreceptors to maintain higher ventilation even during sleep, preventing dangerous nocturnal hypoxaemia.
- ✓ Staged acclimatisation protocol — restrict ascent rate to no more than 300–500 m gain per day above 3,000 m; include rest days to allow 2,3-BPG accumulation, EPO upregulation (takes 3–5 days), and increased ventilation to equilibrate. Educate climbing teams on this schedule before departure from Kampala.
- ✓ Supplemental oxygen delivery — maintain portable oxygen cylinders at Rwenzori base camp (4,800 m) for AMS casualties. Supplemental O2 raises alveolar pO2 back into the range where normal haemoglobin (P50 = 26 mmHg) can load sufficiently, immediately relieving cerebral and pulmonary hypoxia.
- ✓ Immediate descent as priority intervention for severe AMS, HACE, or HAPE — descent of even 300–500 m rapidly increases pO2 and is more effective than any drug intervention for life-threatening high-altitude illness.
- ✓ Monitor with pulse oximetry — SpO2 below 75% at the summit should trigger mandatory rest or descent. Correlates with arterial pO2 data in Table 3; non-acclimatised climbers at 42 mmHg pO2 have SpO2 of approximately 78–80%.

Award max 8. Require: evolutionary mechanism for adaptation (2 marks), minimum 3 distinct clinical protocols with physiological justification (6 marks).

ITEM 3 TOTAL: 20 marks

Part (a) — Neurochemical basis and pharmacological responses (12 marks)

12 marks available

Synaptic transmission — baseline mechanism:

- ✓ Normal synaptic transmission: an action potential arriving at the axon terminal opens voltage-gated Ca2+ channels; Ca2+ influx triggers exocytosis of neurotransmitter-containing vesicles into the synaptic cleft; neurotransmitter binds post-synaptic receptors (ligand-gated ion channels or GPCRs); reuptake transporters and monoamine oxidase (MAO) terminate the signal.

Major Depressive Disorder (MDD):

- ✓ CSF serotonin metabolite (5-HIAA) is severely depleted in MDD (10.2 vs 28.4 ng/mL) — indicates deficient serotonergic neurotransmission in the prefrontal cortex, limbic system, and raphe nuclei. Low serotonin impairs mood regulation, reward processing, and circadian rhythm control.
- ✓ Noradrenaline is also low (188 pg/mL vs 310) — noradrenergic pathways from the locus coeruleus modulate attention, motivation, and energy. Deficiency explains anhedonia, psychomotor retardation, and fatigue characteristic of MDD.
- ✓ SSRIs block the serotonin reuptake transporter (SERT) in the pre-synaptic membrane, preventing serotonin re-entry into the terminal. Synaptic serotonin concentration rises, prolonging post-synaptic receptor activation — this explains 61% symptom reduction in MDD with SSRIs.
- ✓ SSRIs are ineffective for Parkinson's (4% reduction) because Parkinson's is not serotonin-deficient; and only modestly effective in GAD (28%) because GAD's primary deficit is GABAergic, not serotonergic.

Parkinson's Disease:

- ✓ Dopamine metabolite (HVA) is severely depleted (5.4 vs 22.6 ng/mL) — reflects loss of dopaminergic neurons in the substantia nigra (nigrostriatal pathway). Dopamine is essential for initiating and smoothing voluntary movement via the basal ganglia direct/indirect pathway balance.
- ✓ High acetylcholinesterase activity (68.2 vs 42.0 nmol/min/mg) — reflects relative over-activity of cholinergic neurons in the striatum because dopaminergic inhibition of acetylcholine release is lost. Excess acetylcholine contributes to tremor and rigidity.
- ✓ L-DOPA crosses the blood-brain barrier and is decarboxylated to dopamine in residual dopaminergic neurons, directly restoring synaptic dopamine levels and basal ganglia signalling — 72% symptom reduction. L-DOPA is ineffective for MDD and GAD because their pathways are not dopamine-deficient.

Generalised Anxiety Disorder (GAD):

- ✓ GABAa receptor density is severely reduced (62% of control) — GABA is the principal inhibitory neurotransmitter in the CNS. Reduced receptor density means less inhibitory tone in the amygdala and prefrontal cortex, leading to hyperexcitability, persistent worry, and exaggerated fear responses.
- ✓ Noradrenaline is elevated (498 pg/mL vs 310) — excess noradrenergic stimulation of the sympathetic nervous system maintains a chronic fight-or-flight state: tachycardia, muscle tension, and hyperarousal.
- ✓ Benzodiazepines (e.g., diazepam) are positive allosteric modulators of the GABAa receptor — they bind the benzodiazepine site on the receptor, increasing the frequency of Cl- channel

opening in response to GABA. This enhances inhibitory postsynaptic potentials, reducing amygdala and cortical hyperexcitability — hence 68% symptom reduction in GAD.

Award max 12. Require: synaptic baseline mechanism (1), each disorder's neurotransmitter deficit with data reference (6), each drug's mechanism of action with explanation of selectivity (5). Max 9 if no data from Table 4 cited.

Part (b) — Community mental health strategy for Kampala (8 marks)

8 marks available

- ✓ Integrate mental health into Kampala's primary healthcare system — train health centre III/IV staff to administer validated screening tools (PHQ-9 for MDD, GAD-7, UPDRS for Parkinson's early detection), enabling community-level diagnosis without specialist referral, reducing Butabika's patient load.
- ✓ Community pharmacotherapy access — establish a secure supply chain of SSRIs (fluoxetine, affordable generic), L-DOPA/carbidopa, and short-term benzodiazepines at division-level health centres. Carbidopa (peripheral decarboxylase inhibitor) is co-administered with L-DOPA to reduce peripheral conversion, maximising CNS delivery and reducing side effects.
- ✓ Structured psychosocial interventions — deploy Cognitive Behavioural Therapy (CBT) through community health workers trained in low-intensity CBT delivery; CBT has demonstrated efficacy in MDD and GAD that is equivalent to SSRIs in mild-to-moderate cases, without risk of dependence (unlike benzodiazepines).
- ✓ Community exercise programmes — aerobic exercise (150 min/week) increases BDNF (brain-derived neurotrophic factor), promotes neuroplasticity in hippocampal and prefrontal regions, and elevates synaptic monoamine levels independently of drug treatment — evidence-based adjunct therapy for MDD and GAD.
- ✓ Anti-stigma campaigns at parish level — Ugandan sociocultural beliefs often attribute mental illness to spiritual causes, preventing help-seeking. Community radio and drama programmes in Luganda addressing causes and treatability of MDD and anxiety reduce treatment delay, enabling earlier pharmacological and psychosocial intervention when neurochemical deficits are less severe.
- ✓ Parkinson's disease support groups and physiotherapy — tremor and rigidity management with occupational therapy preserves functional independence, reducing caregiver burden; group sessions improve medication adherence and mood in a condition where comorbid depression (MDD + Parkinson's) is common due to dopaminergic/serotonergic dual deficiency.
- ✓ Benzodiazepine prescribing protocol with clear step-down schedule — limit to 2–4 weeks to prevent GABAa receptor downregulation and dependence; transition to SSRIs (effective in 28% of GAD) and CBT for long-term management. Pharmacovigilance committees monitor prescription rates at each health centre.

Award max 8. Require: reference to neurochemical justification for at least 3 strategies (3 marks), distinct pharmacological and non-pharmacological approaches (3 marks), population/public health dimension (2 marks).

ITEM 4 TOTAL: 20 marks

ITEM 5 — Model Answer

AO4 · Ecosystem Ecology, Carbon Cycling & Conservation Biology

AO4 **20 marks**

Part (a) — Ecological and carbon-cycle consequences of deforestation (12 marks)

12 marks available

Shift from carbon sink to carbon source:

- ✓ Intact forest in 1982 was a net carbon sink (−3.8 tCO2/ha/yr): gross photosynthesis by canopy trees exceeded total ecosystem respiration (autotrophic + heterotrophic). Trees fixed CO2 into wood, roots, and soil organic matter faster than decomposers released it.
- ✓ Deforestation removes the photosynthetic biomass — above-ground carbon stocks fell from 210 to 96 tC/ha (a loss of 114 tC/ha over 42 years). Carbon previously locked in wood is released to the atmosphere as CO2 and CH4 through burning (charcoal kilns: 0.1 → 6.2 per km²) and microbial decomposition of felled timber.
- ✓ Soil organic carbon declined from 88 to 44 tC/ha — loss of canopy eliminated leaf litter inputs to soil; exposed soil heats up, accelerating microbial respiration (Q10 effect) and oxidation of humus, releasing stored soil carbon. By 2010 the ecosystem became a net carbon source (+2.1 tCO2/ha/yr), rising to +4.6 by 2024.
- ✓ Data linkage: charcoal kiln density (6.2/km² in 2024) directly correlates with declining carbon stocks and above-source status — each kiln combusts timber, converting solid carbon to atmospheric CO2 and black carbon (soot), which deposits on remaining vegetation and reduces photosynthetic efficiency.

Decline in biodiversity:

- ✓ Tree species richness fell from 148 to 51 per hectare — each species lost eliminates a specific niche (canopy, understorey, root zone), collapsing the three-dimensional habitat structure that vertebrates (birds, mammals) depend on for food, nesting, and shelter. Vertebrate species declined from 312 to 141.
- ✓ Trophic cascade: loss of fruiting tree diversity reduces food availability for frugivorous birds and mammals → primary consumers decline → insectivorous predators that rely on vertebrate prey decline in turn → pollinator populations fall → remaining tree species suffer reduced reproduction → accelerates forest degradation (positive feedback loop).
- ✓ Habitat fragmentation by charcoal clearings creates edge effects — increased wind, temperature, and light penetration at forest edges desiccate interior microhabitats; specialist interior species (obligate forest-interior birds, shade-dependent shrubs) suffer disproportionate decline relative to generalist/edge species.

Decline in rainfall:

- ✓ Annual rainfall in forest plots fell from 1,640 to 1,190 mm — forests generate local precipitation through evapotranspiration: trees pump groundwater to the surface and release it as water vapour via stomata, seeding low-altitude cloud formation. As canopy cover declined from 94% to 53%, evapotranspiration was halved, reducing moisture recycling and local rainfall.
- ✓ Reduced rainfall further inhibits regrowth of cleared areas — creating a drought-deforestation feedback: less water available → slower pioneer plant establishment → slower canopy recovery → continued source status of the ecosystem.

> *Award max 12. Require: carbon sink/source mechanism linked to data (3), deforestation carbon release pathways (2), soil carbon loss mechanism (2), biodiversity trophic cascade (2), rainfall reduction mechanism (2), feedback loop identification (1).*

Part (b) — Restoration and protection framework (8 marks)

8 marks available

- ✓ Immediate legal enforcement of Mabira's reserve boundaries — deploy Uganda Wildlife Authority rangers equipped with GPS mapping tools and camera traps to eliminate illegal charcoal kilns. Each kiln removed reduces CO2 source intensity by approximately 0.74 tCO2/ha/yr (extrapolated from data trend), the single fastest intervention to slow further carbon release.
- ✓ Active reforestation with native pioneer species (Maesopsis eminii, Newtonia buchananii) — pioneers establish quickly, restoring canopy cover; within 10–15 years, canopy cover can return to 65–70%, re-establishing evapotranspiration and rainfall recycling. Target areas with highest soil organic carbon remaining to maximise carbon sequestration rate.
- ✓ Establish a Mabira Biodiversity Corridor connecting the reserve to Namwamba and Kagera forest fragments — allows recolonisation by mobile vertebrates (birds carrying seeds) and mammals, restoring animal-plant mutualistic networks (pollination, seed dispersal) essential for natural regeneration.
- ✓ Community carbon credit scheme — register Mabira under REDD+ (Reducing Emissions from Deforestation and Forest Degradation). Verified carbon sequestration by the restored forest generates internationally tradeable carbon credits; revenue paid to Buikwe district communities as an economic alternative to charcoal, directly addressing the root socioeconomic driver of deforestation.
- ✓ Introduce alternative biomass energy (improved cookstoves, biogas digesters) for communities around Mabira — reduces demand for charcoal by up to 60%; each improved stove substituted reduces one household's contribution to the 6.2 kilns/km² charcoal pressure on the forest.
- ✓ Long-term biodiversity monitoring with permanent study plots — continue the existing Nature Uganda/Makerere monitoring programme with annual censuses of species richness, carbon flux (eddy covariance towers), and canopy cover (satellite imagery). Data-driven adaptive management ensures restoration interventions are adjusted based on measurable outcomes.
- ✓ Advocate for Mabira's inclusion in Uganda's Nationally Determined Contribution (NDC) under the Paris Agreement — committing to its protection provides a binding international obligation, creating legal and political obstacles to future commercial encroachment proposals (as occurred in 2007 when sugarcane expansion was narrowly averted by public protest).

Award max 8. Require: at minimum 4 strategies each with ecological or climate justification. Must include both biodiversity AND carbon/climate angle for full marks. Award 1 bonus mark if REDD+ or NDC mechanism explained correctly.

ITEM 5 TOTAL: 20 marks

ITEM 6 — Model Answer

AO4 · Population Genetics, Evolutionary Biology & Disease Resistance

AO4 **20 marks**

Part (a) — Genetic mechanisms of selective breeding and natural trypanotolerance (12 marks)

12 marks available

Mechanism of resistance allele frequency shift (Gen 0 → Gen 5):

- ✓ In Generation 0, the resistance allele at the TNFRSF13C locus had a population frequency of only 0.12 — most animals were susceptible (homozygous for the susceptibility allele). The programme selectively bred only animals with the highest resistance phenotype (lowest parasite load, highest PCV and survival after tsetse challenge).
- ✓ Directional artificial selection: by choosing only resistant parents for each successive generation, breeders ensured that the resistance allele was consistently over-represented in the gametes contributing to the next generation. Over 5 generations, allele frequency rose from 0.12 to 0.48 — an increase of 0.36, consistent with strong selection coefficients applied each generation.
- ✓ Under Hardy-Weinberg equilibrium (random mating, no selection), allele frequencies remain constant. The observed shift from 0.12 to 0.48 demonstrates a clear departure from Hardy-Weinberg equilibrium, driven by non-random selective mating.

Changes in heterozygosity and inbreeding:

- ✓ Heterozygosity (He) fell from 0.72 to 0.51 — directional selection drives the resistance allele towards fixation, reducing allelic diversity at the selected locus. Simultaneously, the small breeding population used in selection reduces effective population size (Ne: 820 → 290), leading to genetic drift that randomly fixes or eliminates alleles at other loci across the genome.
- ✓ Inbreeding coefficient (F) rose from 0.04 to 0.19 — with Ne of only 290, the probability that two alleles in an individual are identical by descent (IBD) is substantially increased. Inbreeding increases the frequency of homozygous genotypes across all loci, exposing recessive deleterious alleles that were previously masked in heterozygotes — a major long-term genetic risk.
- ✓ Despite increased resistance, parasite load post-challenge (22.6×10^3/mL) remains far above N'Dama levels (5.1×10^3/mL), PCV (28% vs 36%) and survival (67% vs 88%) are also inferior — demonstrating that 5 generations of selection have not achieved the level of trypanotolerance encoded across the N'Dama genome.

Genetic basis of N'Dama natural trypanotolerance:

- ✓ N'Dama cattle exhibit trypanotolerance through naturally high resistance allele frequency (0.81 at TNFRSF13C) accumulated over thousands of years of natural selection in West African tsetse-endemic zones — this is natural selection, not artificial, operating over far more generations.
- ✓ N'Dama's superior tolerance (5.1×10^3/mL parasite load; 88% survival) reflects polygenic resistance — multiple loci across the genome (including MHC class I and II genes controlling T-lymphocyte responses to trypanosomal surface variant antigens) contribute. This contrasts with the single-locus approach of the Soroti programme.
- ✓ N'Dama maintains high heterozygosity (He = 0.68) and low inbreeding (F = 0.06) despite high resistance allele frequency — evidence that natural selection preserves genetic diversity at non-selected loci, maintaining adaptive capacity. The effective population size (740) is 2.5× that of Gen 5 Zebu.
- ✓ Immune mechanism: TNFRSF13C encodes a B-cell activating factor receptor; the resistance allele likely confers more robust humoral immune responses against trypanosomal variant

surface glycoproteins (VSGs), enabling antibody-mediated clearance. N'Dama may also show enhanced innate immune tolerance — reduced inflammatory pathology despite parasitaemia — maintaining body weight (94% vs 68% in Gen 0).

Award max 12. Require: artificial selection mechanism with allele frequency explanation (3), Hardy-Weinberg departure (1), heterozygosity/inbreeding mechanisms with Ne (3), N'Dama polygenic natural selection comparison (3), immune mechanism reference (2).

Part (b) — Long-term genetic risks and sustainable strategy (8 marks)

8 marks available

- ✓ Key long-term risk — inbreeding depression: F = 0.19 in Gen 5 means that approximately 19% of loci are homozygous by descent. Recessive deleterious alleles (for metabolic diseases, skeletal abnormalities, immune deficiencies) are now frequently expressed in homozygous form — reducing fertility, disease resistance to other pathogens, and overall herd productivity. This threatens the commercial viability of the programme.
- ✓ Risk of genetic erosion — effective population size of 290 is below the minimum recommended Ne of 500 for long-term genetic viability. Below Ne = 100 (approaching under continued inbreeding), populations can enter an extinction vortex where inbreeding depression compounds with genetic drift to eliminate beneficial alleles faster than selection can favour them.
- ✓ Introduce rotational crossbreeding with N'Dama — systematic crossing of Gen 5 Zebu resistance lines with N'Dama introduces high-resistance polygenic background, restores heterozygosity, reduces inbreeding coefficient, and imports N'Dama's superior immune gene diversity. F1 crossbreds may exhibit heterosis (hybrid vigour) — exceeding both parental lines in resistance and productivity.
- ✓ Establish a cryopreserved gene bank of semen and embryos from genetically diverse resistant Zebu individuals and N'Dama bulls at NAGRC&DB — preserves allelic diversity permanently, allows restoration of diversity if the live herd suffers an epidemic or bottleneck, and provides material for future genomic selection programmes without further inbreeding.
- ✓ Transition from single-locus selection to genomic selection (GS) — use SNP arrays (50K or 700K bovine SNP chips) to identify and select animals with high genomic estimated breeding values (GEBVs) across multiple resistance loci simultaneously, while applying diversity constraints (penalised relationship matrix) to prevent Ne from falling further.
- ✓ Integrate tsetse vector control with breeding programme — deploy tsetse fly traps, pour-on deltamethrin insecticides, and sterile insect technique (SIT) in Soroti release zones, reducing the infectious challenge intensity. Lower tsetse density reduces selection pressure for extreme resistance, allowing animals with moderate resistance but higher productivity to survive — broadening the viable genetic base.
- ✓ Farmer-participatory breeding programme — involve Soroti pastoralists in selection decisions, balancing trypanotolerance with traits they value (milk yield, draught power, heat tolerance). Local selection criteria ensure the developed breed is actually adopted and maintained rather than abandoned in favour of exotic susceptible breeds if productivity lags.
- ✓ Establish resistance genomics database linking genotype (SNP profiles) to phenotype (parasite load, PCV, survival) across all NAGRC&DB herds — enables marker-assisted selection refinement as new resistance QTLs are identified, and supports international collaboration with ILRI and CIRAD for N'Dama genomic resources.

Award max 8. Require: inbreeding depression risk with mechanistic explanation (2), minimum 3 strategies with genetic or evolutionary justification (5), population genetics principle applied in at least one strategy (1).

ITEM 6 TOTAL: 20 marks