

**SECTION A**  
**ITEM 1**  
**CELLULAR FUNCTIONING**  
**(Compulsory)**

**Areas Assessed**

- + Cytology
- + Histology
- + Chemicals of Life
- + Respiration
- + Nucleic Acids
- + Gene technology
- + Immunity

**Item 1.1**

Mzee Kibuuka, a 45-year-old resident of Lyantonde district in Uganda, has a well-known history of heavy consumption of local potent gins (*tonto* and *waragi*) spanning over fifteen years. Recently, he suffered from a severe, untreated bacterial liver infection.

Following the infection, his health deteriorated rapidly. He developed several visible complications, including jaundice (yellowing of the eyes and skin), ascites (severe swelling of the abdomen due to fluid retention), extreme fatigue, and persistent bleeding from minor cuts.

Concerned by his condition, his family took him to Mulago National Referral Hospital. A gastroenterology specialist performed a liver biopsy and blood tests.

The specialist's diagnostic report compared Mzee Kibuuka's liver tissue samples with those of a healthy individual. Additionally, routine blood work revealed that Mzee Kibuuka had abnormally high blood glucose levels (hyperglycemia).

Cellular / Blood Parameter	Normal Individual's Liver Tissue	Mzee Kibuuka's Liver Tissue
Cell Membrane Structure	Intact, selectively permeable, distinct phospholipid bilayer.	Fragmented, highly porous, and leaky bilayer.
Nucleus & DNA	Intact nuclear envelope; active transcription of metabolic enzymes.	Ruptured nuclear membrane; fragmented DNA; halted transcription.
Endoplasmic Reticulum (ER)	Extensive rough ER (high ribosome density) and smooth ER.	Severe de-granulation (loss of ribosomes); dilated and collapsed tubules.
Golgi Apparatus	Well-developed, stacked cisternae actively packaging proteins.	Flattened cisternae dispersed and structurally degraded.
Blood Glucose Level	Normal fasting range (3.9 - 5.6 mmol/L).	Elevated fasting level (14.2 mmol/L).

**Task**

- a) Analyze the data and provide a suitable explanation of how the destruction of the specified cellular structures affects overall cellular and body functions.
- b) Describe how Mzee Kibuuka's Liver Tissue would regulate increase in the electrolyte before the infection?
- c) Provide suitable, practical strategies that can be implemented to manage Mzee Kibuuka's health challenge.

**KABALEGA SS**

### Item 1.2

Okello, a 28-year-old youth living in a suburb of Kampala, has been struggling with intravenous (IV) drug addiction for three years, frequently sharing unsterilized needles. He was recently rushed to Kiruddu National Referral Hospital presenting with a high persistent fever, severe shortness of breath during minimal exertion (dyspnea), swollen legs and ankles (peripheral edema), and regular fainting spells (syncope).

The cardiologist diagnosed him with **Infective Endocarditis**. This is a severe microbial infection of the inner lining of the heart (*endocardium*) and its valves, frequently caused by bacteria introduced directly into the bloodstream via dirty needles. The infection triggers inflammatory lesions called *vegetations* that physically destroy heart tissue and valve structures. Furthermore, the infection spread into the myocardium (the heart muscle layer) and the specialized electrical pathway running through the cardiac septum, causing a severe **delay in his heartbeat (heart block)**.

The specialist compiled a report comparing Okello's cardiovascular function and cardiac cell tissue with that of a healthy individual:

Parameter Evaluated	Normal Individual	Okello's Medical Report
Gross clinical complications	Efficient blood circulation; no fluid retention; normal breathing.	Severe backflow of blood (regurgitation); fluid accumulation in lungs and tissues; poor tissue perfusion.
Electrical pulse conduction	Rapid propagation via SA node, AV node, and Purkinje fibers. Rate: 60-100 bpm.	Extended PR interval / delayed signal travel across the fibrous septum. Rate: 38 bpm (Bradycardia).
Mitochondria (Cardiac Cell)	High density; dense, intact folded inner membranes (cristae).	Swollen, ruptured outer membranes; completely disrupted and lost cristae.
Gap junctions & intercalated discs	Intact, tightly interlocking protein channels between cardiac cells.	Completely degraded intercalated discs; disconnected cell-to-cell channels.
Sarcoplasmic reticulum (sr)	Well-organized network; active calcium storage and release.	Fragmented, dilated tubules with severely impaired calcium binding.

#### Task

- Analyze the data to provide a suitable explanation of how the disease affected the cardiac tissue and how the subsequent clinical complications arose.
- Describe how Okello's heart would work under normal conditions before developing the complication.
- Provide suitable, practical strategies that can be implemented to manage Okello's health challenge.

**GREEN FIELD SS**

### Item 1.3

Opio, a 32-year-old casual laborer in Gulu City, Uganda, has a documented history of chronic intravenous (IV) heroin and contaminated chemical drug abuse. Recently, he developed a severe microbial kidney infection (*pyelonephritis*) directly introduced via unsterile needles, which went untreated for weeks due to his fear of stigmatization.

Opio was rushed to Gulu Regional Referral Hospital after collapsing. He presented with severe clinical complications: minimal, dark-colored urine production (**oliguria**), massive swelling of his entire face and limbs (**generalized edema**), and extremely high blood pressure (**hypertension**).

The nephrology specialist diagnosed Opio with **Acute Tubular Necrosis (ATN) and Glomerulonephritis** a severe medical complication involving the profound destruction of the functional filtration and reabsorption tissues of the kidney (the nephrons). The expert medical report revealed that toxic drug metabolites combined with the bacterial infection destroyed his kidney tissues, causing an abnormal accumulation of sodium ions ( $\text{Na}^+$ ) in his blood and a complete failure of **osmoregulation**.

Kidney Tissue Component	Healthy Tissue Structure (Before)	Opio's Tissue Structure (After Diagnosis)
Glomerulus & podocytes	Intact endothelial pores; healthy podocyte slit diaphragms for selective ultrafiltration.	Severely inflamed scarred capillaries; flattened and destroyed podocyte filtration slits.
Proximal convoluted tubule	Tall cuboidal epithelial cells with dense microvilli (brush border) and high mitochondrial density.	Flattened, dead epithelial cells ( <i>necrosis</i> ); complete loss of microvilli brush border.
Collecting duct cells	Intact aquaporin water channel proteins; responsive to Antidiuretic Hormone (ADH).	Sloughed-off epithelial lining; damaged, non-functional aquaporins; unresponsive to ADH.

**Table 3: Changes in Kidney Tissue Structures**

Parameter/Complication	Normal Value (Healthy Individual)	Opio's Diagnostic Values
Blood Sodium Ion Level	135 - 145mmol/L	<b>Elevated:</b> 168mmol/L (Severe Hyponatremia)
Blood Osmolarity	275 - 295mOsm/kg	<b>Elevated:</b> 340mOsm/kg (Highly concentrated blood)
Fluid Volume in Tissues	Normal interstitial balance (no swelling).	<b>Severe Edema:</b> Massive water retention in tissues.
Blood Pressure	120 / 80mmHg	<b>Severe Hypertension:</b> 180 / 110 mmHg
Urine Output	Approximately 1.5Liters / day	<b>Oliguria:</b> Less than 0.3Liters / day.

**Table 4: Changes in Homeostatic Parameters & Clinical Complications**

#### Task

a) Analyze the data and provide a suitable explanation of how the disease affected the tissues and how his clinical complications came about.

b) Describe how his body would normally regulate sodium ions and maintain osmoregulation before he developed this condition.

c) Provide suitable, practical strategies that can be implemented to manage Opio's health challenge in Uganda.

**KABALEGA SS**

**Item 1.3**

In **Tiira Village, Busia District, Uganda**, a resident accidentally left out a local paste mixed with an unauthorized, cheap chemical rat poison containing sodium cyanide. The couple's 4-year-old child discovered and ingested a small portion of the food. Within minutes, the child presented with acute respiratory distress, severe dizziness, confusion, and vomiting, before collapsing. The child was rushed to **Busia Health Centre IV**. Upon admission, the clinical team noted that this emergency mirrored a cluster of admissions reported earlier that month at the same facility. Those previous cases involved small-scale artisanal gold miners and local community members residing along the banks of the nearby **River Namukobe**. An upstream processing plant had discharged untreated industrial effluent containing active cyanide complexes directly into the river water.

To confirm the underlying pathophysiology, the consulting toxicologist ordered immediate arterial blood gas tests, metabolic panels, and muscle biopsy sub-cellular assays. The doctor compiled the results into a comparative data profile against a healthy individual from the same community. The raw data was presented to the medical board without an interpretive narrative but indicated that Cyanide acts as a classic **non-competitive inhibitor** of **cytochrome c oxidase**.

Parameter Measured	Healthy Individual (Control)	Cyanide-Exposed Patient
Arterial Blood O <sub>2</sub> Saturation	98%	97%
Venous Blood O <sub>2</sub> Saturation	70%	93%
Blood Lactate Concentration	1.0mmol/L	11.5mmol/L
Arterial Blood PH	7.40	7.12
Intracellular ATP Production Rate	38 molecules per Glucose	2 molecules per Glucose
Mitochondrial Oxygen Consumption Rate	High	Near Zero
Citric Acid (Krebs) Cycle Activity	Normal / Active	Inhibited
Rate of Glycolysis	Normal / Baseline	Significantly Accelerated
Core Body Organismal Signs	Normal breathing pulse	Severe hyperventilation, tachycardia

**Table 1: Cellular and Organismal Physiological Parameters**

**Tasks:**

a) Analyze the clinical and cellular data provided in Table 1 and explain the differences between the healthy individual and the cyanide-exposed patient.

b) Based on the scenario provided, suggest and justify suitable, actionable strategies to manage and prevent this challenge at two distinct levels. **GREEN FIELD**

**Item 1.4**

Juma, a 54-year-old commercial motorcycle (*boda-boda*) rider, lives and operates a small kiosk along a busy, unpaved murram road in Wakiso District, Uganda. The road is notoriously dusty, continually stirred up by heavy traffic. In addition to inhaling thick dust daily for over twenty years, Juma has been a heavy chronic smoker, consuming a pack of commercial cigarettes daily since his youth.

Recently, Juma’s health deteriorated significantly. He developed severe clinical complications, including a persistent, hacking cough that produces thick mucus, extreme shortness of breath (dyspnea) even while sitting, and a noticeably chest-heavy, rapid breathing rate.

Alarmed by his constant exhaustion, his family took him to the Uganda Heart Institute and the respiratory unit at Mulago National Referral Hospital. Clinical assessments revealed profound structural destruction within his respiratory tract, causing severe secondary complications in his circulatory system specifically pulmonary hypertension (abnormally high blood pressure in the lung arteries) and right-sided heart failure (cor pulmonale) due to the heart straining to pump blood through damaged lung tissue.

The specialist diagnosed Juma with Severe Emphysema and Chronic Bronchitis (collectively known as Chronic Obstructive Pulmonary Disease, or COPD). The expert diagnostic data compares Juma's lung tissues with those of a healthy, non-smoking individual:

<b>Lung Tissue Component</b>	<b>Healthy Tissue Structure</b>	<b>Juma’s Tissue Structure (After Diagnosis)</b>
Alveolar walls and surface area	Millions of tiny, distinct, elastic sacs providing a massive surface area for gas exchange.	Alveolar walls broken down and merged into fewer, large, sagging sacs; total surface area drastically reduced.
Elastic fibers and elastin	High density of elastin fibers; allows lungs to recoil passively during exhalation.	Severe destruction of elastin fibers; lungs lose elasticity and remain permanently over-inflated.
Ciliated epithelium and goblet cells	Healthy, moving cilia rows; normal goblet cells producing a thin, protective layer of mucus.	Cilia completely paralyzed and destroyed; goblet cells enlarged (hypertrophy), producing excess thick mucus.
Pulmonary capillary network	Dense, rich network of capillaries tightly wrapping around every alveolus.	Capillary beds compressed, damaged, and drastically reduced in number due to alveolar collapse.

**Table 1: Changes in Respiratory Tissue Structures**

**Task**

a) Analyze the data and provide a suitable explanation of how the condition affects the functioning of the lung tissues and how the subsequent respiratory and circulatory complications arose.

b) Evaluate Juma's condition and provide suitable, practical strategies that can be implemented in a Ugandan context to manage this health challenge.

KABALEGA

**Item 1.5**

During the **Uganda Interdistrict Football Competitions**, a star midfielder from the local district team was traveling by bus to the regional finals. While passing through a busy trading center, the team bus pulled over for a brief rest break. The athlete rushed to a roadside canteen located adjacent to a commercial chemical processing factory—a facility that local residents had repeatedly petitioned local authorities about for dumping industrial effluents into the local river used by the community for cooking and watering livestock. The athlete bought and drank a large glass of fresh local milk.

Within thirty minutes of returning to the bus, the athlete began experiencing severe physiological symptoms of acute toxin exposure: intense muscle fatigue, severe abdominal cramps, persistent nausea, extreme dizziness, a rapid heart rate (tachycardia), and labored, rapid breathing (hyperventilation), culminating in a sudden physical collapse. The athlete was rushed to the emergency unit of the nearest **Health Centre IV**.

Upon clinical diagnosis, the medical toxicologists confirmed that the milk was contaminated with **fluoroacetate**, a notorious metabolic poison originating from the factory’s poorly managed waste. The doctors stated that once inside the cells, fluoroacetate is metabolized into **fluorocitrate**, a toxic analog that acts as a potent biochemical inhibitor of **aconitase** (aconitate hydratase) a key regulatory enzyme in the **Citric Acid (Krebs) Cycle**.

To evaluate the clinical interns, the senior consultant compiled the lab findings comparing the poisoned athlete against a healthy, unexposed teammate (control) into a comparative profile without any explanatory narrative.

Parameter Measured	Healthy Teammate (Normal Control)	Fluoroacetate-Exposed Athlete
Blood Citrate Accumulation Rate	Normal / Baseline	Extremely High (10× elevation)
Isocitrate & Alpha-Ketoglutarate Levels	Normal / Steady State	Depleted / Near Zero
Mitochondrial NADH/FADH <sub>2</sub> Production	High / Optimal	Drastically Reduced
Intracellular ATP Production Yield	38 molecules per Glucose	2 molecules per Glucose
Mitochondrial Oxygen Consumption Rate	High / Active	Severely Depressed
Rate of Cellular Glycolysis	Normal / Baseline	Significantly Accelerated

Blood Lactate Concentration	0.9 mmol/L	12.5 mmol/L
Arterial Blood pH	7.40	7.11
Core Organismal Status	Peak athletic stamina	Severe hyperventilation, muscle rigidity, collapse

Table 2: Cellular, Metabolic, and Organismal Physiological Parameters

**Task**

- Analyze the clinical and cellular data provided in Table 2 and provide a suitable explanation on the difference in parameters between the two individuals.
- Describe how the enzyme would be able to work and enable him perform favourably in the competition if he had not consumed the poisoned milk?
- Provide suitable strategies that can be implemented to manage the challenge.

GREEN FIELD

**Item 1.6**

During the **Uganda National Secondary Schools Athletics Championships** held at the Mandela National Stadium, Namboole, spectators witnessed an intriguing phenomenon during the Men's 400-metre final. All eight competing athletes exploded out of the starting blocks at an exceptionally high speed. However, sports science students in the stands noticed that after the first 50 to 80 metres, the athletes' speeds progressively and visibly reduced as they pushed through the backstretch and home straight, despite executing maximum physical effort.

Among the competitors was an athlete from the West Nile region who had recently been battling severe **iron-deficiency anaemia** and a chronic **respiratory tract illness**. Although he started at a similar explosive pace out of the blocks, his speed dropped drastically earlier than the others, and his overall performance was significantly lower, causing him to finish last with profound physical exhaustion and muscle rigidity.

The Uganda Olympic Committee medical team tracked the physiological profiles of the athletes. Using wearable biosensors and immediate post-race blood sampling, they compiled data from a typical healthy finalist and the recently ill, anaemic athlete. The parameters were measured at three intervals: **Before the race (Resting)**, **During the race (at the 250m mark)**, and **5 minutes After the race (Recovery)**.

Parameter Measured	Athlete Condition	Before the Race (Resting)	During the Race (250m Mark)	5 Mins After the Race (Recovery)
Muscle Creatine Phosphate (CP) concentration	Healthy Anaemic	100% 100%	12% 8%	65% 30%
Blood Lactic Acid concentration	Healthy Anaemic	1.0 mmol/L 1.2mmol/L	11.8 mmol/L 18.5mmol/L	14.2 mmol/L 21.0mmol/L
Arterial Blood Oxygen Saturation (SaO <sub>2</sub> )	Healthy Anaemic	98% 92%	95% 81%	97% 89%

Ventilation Rate (Breaths per minute)	Healthy Anaemic	14 16	48 62	32 45
Cardiac Output (Litres of blood pumped/min)	Healthy Anaemic	5.0L 5.5L	22.0 L 24.5L	12.0L 16.0L
Core Organismal Status & Speed	Healthy  Anaemic	Calm, normal  Slightly fatigued	Fast start, gradual slowdown  Severe deceleration, heavy gasping	Gradual recovery  Persistent muscle cramps, hyperventilation

**Table 1: Physiological and Metabolic Profiles of the Athletes**

**Task**

- Analyse the data and provide a suitable explanation on the observed difference in performance during the race.
- Evaluate the information provided and provide suitable strategies that can be implemented to manage the challenge.

**KABALEGA**

**SECTION A**

**ITEM 2**

**PLANT ANATOMY AND PHYSIOLOGY**

**(Compulsory)**

**Areas Assessed**

- + Nutrition in Plants
- + Gene Technology
- + Homeostasis in Plants
- + Coordination in Plants
- + Growth in Plants and Development in Insects

**2.1**

Farmers at the Lake Victoria lowlands in Kasensero, Kyotera District, have been practicing a mixed-cropping system, planting maize (*Zea mays*) and beans (*Phaseolus vulgaris*) concurrently in the same fields. Over the last five consecutive farming seasons, the farmers noticed a frustrating trend: while their maize crops flourished and produced consistently high grain yields, the bean crops remained stunted, suffered from visible leaf scorching, and produced very low pod yields. The local sub-county agricultural extension officer visited the farms to investigate. After evaluating the climate characterized by high ambient temperatures, intense tropical solar radiation, and periods of prolonged afternoon sun—the officer took leaf samples to the laboratory. The officer attributed the drastic yield differences to fundamental differences in the internal leaf anatomy and photosynthetic pathways of the two crops, noting that the hot, open lowlands favor one over the other.

The laboratory analysis revealed clear structural and functional differences between the leaves of the two crops:

Anatomical / Physiological Parameter	Maize Leaf ( <i>Zea mays</i> )	Bean Leaf ( <i>Phaseolus vulgaris</i> )
Photosynthetic Pathway	C4 Pathway (Hatch-Slack Pathway).	C3 Pathway (Calvin Cycle alone).
Internal Leaf anatomy	Kranz Anatomy present: Concentric rings of large, thick-walled bundle sheath cells surrounding the vascular bundles, tightly wrapped by mesophyll cells.	Standard Anatomy: Differentiated into upper palisade mesophyll and lower spongy mesophyll; no specialized bundle sheath cells.
Primary Carbon-Fixing Enzyme	PEP Carboxylase (located in mesophyll cells); has zero affinity for oxygen (O <sub>2</sub> ).	RuBisCO (located throughout the mesophyll); has a high affinity for both CO <sub>2</sub> and O <sub>2</sub> .
Optimal Temperature Range	High temperatures (30°C – 45°C).	Moderate temperatures (15°C – 25°C).
Photorespiration Rate	Negligible / Absent.	High (especially during hot, bright days).

### Task

- Analyze the data and provide a suitable explanation of how the differences in leaf anatomy and structure are directly related to the differences in crop yields at the Lake Victoria lowlands.
- Evaluate the environmental conditions and provide suitable, practical strategies that the farmers can implement to manage the challenge and ensure improved bean yields in subsequent seasons.

### KABALEGA

#### Item 2.2

In the high-altitude volcanic slopes of Kapchorwa District, located within the Sebei sub-region of Eastern Uganda, farmers practice mixed cropping by planting sorghum (*Sorghum bicolor*) and beans (*Phaseolus vulgaris*) side by side on their terraced fields. Over the past few years, farmers have consistently observed a perplexing trend: their bean crops thrive exceptionally well, producing abundant, heavy pod yields, whereas their sorghum crops remain severely stunted, exhibit pale green leaves, and produce very poor grain yields.

The district agricultural extension officer visited the high-altitude farms to assess the situation. The officer noted that Kapchorwa is characterized by cool ambient temperatures (often falling below (15°C – 18°C), high misty humidity, and reduced light intensity due to frequent cloud cover. The officer collected leaf samples from both crops and took them to the laboratory for analysis. The diagnostic report revealed that the yield disparity is caused by fundamental differences in the internal leaf anatomy and photosynthetic biochemical pathways of the two crops, noting that the montane highland microclimate strongly discriminates against one of them. The laboratory analysis compared the structural and functional features of the leaves of both crops:

Parameter	Sorghum Leaf ( <i>Sorghum bicolor</i> )	Bean Leaf ( <i>Phaseolus vulgaris</i> )
Photosynthetic Pathway	C4 Pathway (Hatch-Slack Pathway).	C3 Pathway (Calvin Cycle alone).
Internal Leaf Anatomy	Kranz Anatomy present: Concentric rings of large, thick-walled bundle sheath cells tightly wrapped by mesophyll cells.	Standard Anatomy: Differentiated into distinct upper palisade mesophyll and lower spongy mesophyll; no specialized bundle sheath cells.
Primary Carbon-Fixing Enzyme	PEP Carboxylase (located in the mesophyll cells).	RuBisCO (located throughout the open mesophyll layers).
Enzyme optimum temperature	High temperatures (30°C – 45°C). High sensitivity to cold.	Cool to moderate temperatures (15°C– 25°C).
Photorespiration Rate	Negligible / Suppressed.	Negligible in cool conditions; high only under heat.

### Task

- Analyze the data and provide a suitable explanation of how the differences in leaf anatomy and structure are directly related to the differences in crop yields in the Kapchorwa highlands.
- Evaluate the environmental conditions and provide suitable, practical strategies that the farmers can implement to manage the challenge and ensure improved sorghum yields in subsequent seasons.

### GREEN FIELD

### Item 2.3

Mr. Orombi, a progressive smallholder farmer in the dry, semi-arid plains of the Terego district within the **West Nile sub-region**, recently expanded his cultivation of UGPMA1 (an improved open-pollinated maize variety). During the prolonged dry spell of the first planting season, his crop showed severe signs of moisture stress, including stunted growth, significant leaf rolling, and localized wilting.

Concerned about the impending crop failure, crop physiologists from the **Ministry of Agriculture, Animal Industry and Fisheries (MAAIF)** visited Mr. Orombi's farm to audit the physiological health of the stressed crops. The analysts gathered biochemical and environmental data from the affected fields and compared them with data from a fully irrigated control plot at a nearby research station.

The expert data compiled by the Ministry analysts is presented in the two data sets below:

Hormone Type	Irrigated Control Plot (ng/g Fresh Weight)	Mr. Orombi's Stressed Field (ng/g Fresh Weight)
Abscisic Acid	25.2	185.4
Gibberellins	14.8	3.1
Auxins (IAA)	32.5	8.7

**Table 1: Endogenous Phytohormone Levels in Maize Leaves**

Parameter Assessed	Mr. Orombi's stressed field	Irrigated Control Plot
Stomatal Conductance ( $\text{gs} \cdot \text{mmolm}^{-2} \cdot \text{s}^{-1}$ )	45	320
Intercellular $\text{CO}_2$ Concentration ( $\text{Ci}$ , ppm)	110	280
Net Photosynthetic Rate	6.2	28.5
Chlorophyll Fluorescence Ratio	0.58	0.82

**Table 2: Photosynthetic and Physiological Parameters**

(a) Analyze the biochemical and physiological data provided by the MAAIF analysts. Provide a suitable, detailed scientific explanation for the physical changes observed in Mr. Orombi's maize crops.

b) Evaluate the sustainable scientific measures and agronomic interventions that Mr. Orombi can apply to solve these physiological limitations and improve overall crop productivity in the West Nile region.

### GREEN FIELD

#### Item 2.4

Mr. Kiptoyek, a commercial farmer in the high-altitude, cool highlands of the **Kapchorwa district** on the slopes of Mt. Elgon, decided to plant a high-yielding variety of Maize (*Zea mays*), which is a **C4 plant**. Despite using recommended nitrogenous fertilizers and ensuring adequate rainfall, Mr. Kiptoyek consistently attained drastically lower grain yields compared to farmers growing the same variety in the warmer, low-altitude districts of Serere and Soroti.

Believing the issue was premature leaf senescence (aging) and poor grain filling, Mr. Kiptoyek heavily sprayed his crop last season with a commercial foliar fertilizer enriched with **cytokinins** (hormones that promote cell division and delay leaf aging). However, his harvest only showed a marginal increase, and overall yields remained relatively low.

Crop scientists from the National Agricultural Research Organisation (NARO) visited the farm and conducted a comprehensive physiological audit. They compared Mr. Kiptoyek's high-altitude maize crop with a control plot of the same maize variety grown at its optimal, warm low-altitude research station.

The expert data compiled by the NARO scientists is presented in the table below:

Parameter Assessed	Optimal Low-Altitude Station (Warm)	Mr. Kiptoyek's Farm (High-Altitude, Cool)
Average Growing Temperature ( $^{\circ}\text{C}$ )	28.5	14.2
Kranz Anatomy Development	Fully developed bundle sheath cells	Poorly developed, thin-walled bundle sheath cells
Activity of PEP Carboxylase Enzyme ( $\mu\text{molmg}^{-1} \text{proteinmin}^{-1}$ )	4.5	0.8
Endogenous Cytokinin levels in Grain ( $\text{ng/g}$ Fresh Weight)	42.0	11.5
Net Photosynthetic Rate ( $\text{Pnm}, \mu\text{molCO}_2 \text{m}^{-2} \text{s}^{-1}$ )	38.2	12.4
Rate of Photorespiration (% of total fixed Carbon)	Less than 2%	18%

**Table 1: Anatomical, Hormonal, and Photosynthetic Parameters of the Maize Crop**

### Tasks

- Analyze the data provided in the scenario and Table 1. Provide a suitable, detailed physiological explanation for the low yields continuously obtained by Mr. Kiptoyek despite the high altitude.
- Evaluate the cytokinin foliar spraying strategy implemented by the farmer, and provide other suitable agronomic or biological strategies he can implement to improve crop yields in Kapchorwa.

### KABALEGA SS

#### Item 2.5

Okello is a small-scale commercial farmer in Nakasongola district—a semi-arid area located within Uganda’s cattle corridor. Hoping to supply the nearby urban centers, Okello planted two acres of high-value crops: exotic flowers and fast-growing vegetables (tomatoes and cabbages). Lately, prolonged dry spells have hit his farm hard. The soil has dried up and cracked, causing severe **water stress** to the crops. Okello noticed that his plants are showing clear signs of drought damage: extensive **leaf wilting**, **stunted growth**, premature **dropping of flower buds (abscission)**, and yellowing of older leaves (**chlorosis**) due to poor nutrient uptake from the dry ground. Desperate to save his investment, he consulted an agricultural expert from the Ministry of Agriculture, Animal Industry and Fisheries (MAAIF).

The expert explained that under drought stress, plants naturally reduce production of growth-stimulating hormones and increase stress hormones like Abscisic Acid (ABA), which halts development. The expert advised Okello to spray his crops with a bio-stimulant liquid fertilizer enriched with two key plant hormones: **cytokinins** and **auxins**. Okello applied the hormonal spray as a fine mist during the cool early morning hours.

The agricultural extension team monitored Okello’s farm and recorded the following average plant metrics across his plot 2 weeks *before* the application and 4 weeks *after* the continuous application of the hormonal spray.

Plant Physiological Parameter Measured	Before Hormone Spray Application	After Hormone Spray Application
Average Root Depth (cm)	14.5 cm	31.2 cm
Stomatal Conductance ( $\text{mmol m}^{-2}\text{s}^{-1}$ ) (Rate of stomata opening)	45.0 (Stomata largely closed)	185.0 (Stomata moderately open)
Chlorophyll Content Index (CCI)	18.2 (Pale yellow-green leaves)	42.5 (Deep green leaves)
Flower Bud Abscission Rate (%)	72% loss (Severe shedding)	14% loss (High retention)
Average Crop Yield per Acre (kg)	320 kg	1,450 kg

## Tasks

- Analyze the data provided in the table. Provide a comprehensive biological explanation of how the application of cytokinins and auxins helped Okello's crops overcome the drought-related effects to improve yield.
- Evaluate the environmental and climatic conditions in Nakasongola district. Suggest and justify three sustainable agronomic strategies (other than chemical spraying) that Okello can implement to permanently maintain high yields of flowers and vegetables under semi-arid constraints.

**GREEN FIELD SS**

**SECTION B**  
**PART I**  
**ITEM 3& 4**  
**HUMAN PHYSIOLOGY**  
**(Attempt Only One Item)**

### Areas Assessed

- ✚ Transport in Animals
- ✚ Respiration
- ✚ Homeostasis in Animals
- ✚ Coordination in Animals and Behavior

### Item 3.1

Two middle-distance runners, **Athlete A (Healthy/Control)** and **Athlete B**, were scheduled to compete in a 10,000meter training race.

Two days before the race, **Athlete B** made a severe mistake: he slept in a small, poorly ventilated room with a burning charcoal stove (*sigiri*) to keep warm during a cold night. He woke up with severe headaches, dizziness, and nausea—classic symptoms of acute **carbon monoxide (CO) poisoning**. Furthermore, Athlete B's recent lifestyle choice aggravated his condition; he had been strictly observing a religious Lent fasting period, which was coupled with a poor feeding habits lacking in essential green leafy vegetables and iron-rich foods, leading to a clinical diagnosis of **iron-deficiency anemia**.

On the day of the race, both athletes ran under identical environmental conditions. While Athlete A maintained a steady pace and recovered quickly, Athlete B struggled significantly. He experienced intense muscle fatigue, hyperventilation (rapid breathing), a dangerously elevated heart rate, and localized muscle cramping, ultimately finishing long after Athlete A.

Medical personnel collected blood and circulatory metrics from both athletes 30 minutes **before the race** (at rest) and 10 minutes **after the race** (post-exertion).

Physiological Parameter Measured	Athlete A (Healthy) Before → After	Athlete B (Anemic + CO Poisoned) Before → After
Blood Carboxyhemoglobin (COHb) Level (%)	0.5% → 0.5%	28.0% → 26.5%
Total Functional Hemoglobin Count (g/dL)	15.2 → 15.2	9.1 → 9.1
Cardiac Output (L/min) ( <i>Volume of blood</i> )	5.0 → 22.0	7.5 → 32.0

Physiological Parameter Measured	Athlete A (Healthy) Before → After	Athlete B (Anemic + CO Poisoned) Before → After
<i>pumped/min)</i>		
Blood Lactic Acid Concentration (mmol/L)	1.2 → 4.5	3.8 → 16.2
White Blood Cell (WBC) Count (cells/ $\mu$ L)	6,000 → 9,500	11,500 → 18,000

### Task

- Analyze the data provided in the table. For each of the five physiological parameters, explicitly state the trend comparing Athlete A to Athlete B (both at rest and after the race) and provide a comprehensive biological explanation of how Athlete B's conditions affected his performance.
- Evaluate the biological failures hindering Athlete B's performance. Suggest and justify four distinct, scientifically sound strategies that Athlete B must implement during or after his fasting periods to fix his internal physiology, recover his oxygen-carrying capacity, and safely maintain high athletic performance.

### GREEN FIELD S.S

### Item 3.2

Atim, a 28-year-old mother residing in Gulu District, northern Uganda, attended her first antenatal care (ANC) visit at Gulu Regional Referral Hospital during her second pregnancy. Her first child had been delivered smoothly two years prior at a local health center without any medical complications and is currently healthy.

However, during this second pregnancy, routine fetal ultrasounds and subsequent post-delivery observations revealed severe clinical complications in the newborn. The second baby was born prematurely, showing severe yellowing of the skin and eyes (**jaundice**), extreme weakness due to a massive destruction of red blood cells (**severe hemolytic anemia**), and generalized body swelling (**hydrops fetalis**).

The doctors immediately initiated emergency treatment, including an **exchange blood transfusion** and intensive **phototherapy** to save the baby's life. Following successful recovery, and as part of the Uganda National Expanded Programme on Immunisation (UNEPI) catch-up schedule, the child was also administered a live-attenuated **measles vaccine**.

During management, the medical laboratory team collected blood samples to analyze specific immune indicators in both Atim's (maternal) circulation and the baby's (fetal) circulation. The data is presented in the table below

**Table 3.1: Concentration of Key Immune Indicators in Maternal and Fetal Circulation**

<b>Immune Indicator Analyzed</b>	<b>Maternal Circulation (Atim)</b>	<b>Fetal Circulation (First Born)</b>	<b>Fetal Circulation (Second Born)</b>
<b>Rhesus (D) Antigen Presence</b>	Absent (Rh <sup>-</sup> )	Present (Rh <sup>+</sup> )	Present (Rh <sup>+</sup> )
<b>Anti-Rhesus (Anti-D) IgM Antibodies (g/L)</b>	2.4	0.0	0.0
<b>Anti-Rhesus (Anti-D) IgG Antibodies (g/L)</b>	8.5	0.0	7.8
<b>Total Red Blood Cell Count (times 10<sup>12</sup>L)</b>	4.2	5.1	1.8




**Tasks**

- Analyse the scenario and data provided in Table 1. Account for the trend/differences observed in the immune indicators and red blood cell counts between the mother and the second-born child.
- Explain why the second-born child suffered severe complications, yet the first-born child did not face any complications at all.
- Explain why the doctors offered a measles vaccine to the child after treatment and describe how it boosts the baby's long-term immunity against measles.
- Provide well-justified medical strategies on how the child's life-threatening condition can be completely prevented in Atim's subsequent pregnancies.

**KABALEGA S.S**

**SECTION B  
PART II  
ITEM 5& 6  
(Attempt Only One Item)**

**Areas Assessed**

-  Variation
-  Genetics
-  Evolution
-  Ecology