

HOLIDAY PROGRAM: BIOCLAS A-LEVEL BIOLOGY

DAILY TRIALS

In Jinja Industrial Area, workers in a beverage processing factory reported increasing cases of fatigue, muscle weakness, and slow wound healing.

The factory uses high temperatures and chemical preservatives during production. Workers are exposed to fumes and long working hours.

Observations showed:

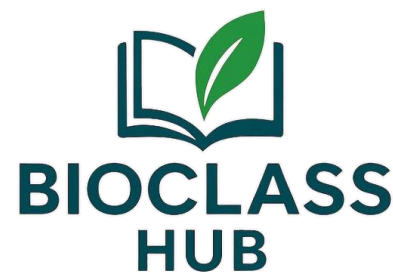
- Some workers frequently skip meals during long shifts
- Safety masks are available but not consistently used
- Workers handle enzymes used in industrial processing
- Some workers rarely take rest breaks
- Waste chemicals are discharged into nearby drainage channels
- A few workers regularly go for medical check-ups

Microscopic examination of muscle tissue samples revealed:

- Reduced mitochondrial density
- Damaged cell membranes
- Low enzyme activity

Table 1: Cellular and Respiratory Indicators

Parameter	Healthy Worker	Exposed Worker
ATP production rate (%)	100	50
Enzyme activity (%)	100	40
Oxygen consumption rate	Normal	Low
Mitochondrial number	High	Reduced
Membrane integrity (%)	Intact	Damaged
Muscle contraction strength (%)	High	Low



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Task

- (a) Analyse how enzyme inhibition, mitochondrial damage, and membrane disruption reduce oxygen utilisation, ATP production, and muscle performance in workers.
- (b) Propose practical measures that would improve cellular function, restore energy production, and reduce harmful exposure.

Enzyme inhibition by industrial chemicals reduces active site availability through competitive and non-competitive mechanisms; altered enzyme-substrate specificity lowers catalytic efficiency in pathways such as **glycolysis (hexokinase, phosphofruktokinase)** and the **Krebs cycle (dehydrogenases)**, decreasing formation of reduced coenzymes (**NADH, FADH₂**), limiting electron supply to the **electron transport chain**, reducing proton gradient formation across the inner mitochondrial membrane and lowering **ATP synthesis by ATP synthase**, resulting in reduced cellular energy.

Mitochondrial damage reduces inner membrane surface area (cristae) where the **electron transport chain (cytochromes, NADH dehydrogenase, cytochrome oxidase)** operates; impaired electron transfer decreases proton pumping, collapsing the electrochemical gradient required for **chemiosmosis**, lowering oxidative phosphorylation efficiency and ATP yield, leading to insufficient energy for muscle contraction.

Reduced oxygen utilisation limits the final electron acceptor in the electron transport chain; electrons accumulate in reduced carriers, slowing **cytochrome c oxidase activity**, reducing water formation and halting efficient electron flow, decreasing ATP production and forcing reliance on anaerobic pathways with low energy yield.

Membrane damage (fluid-mosaic disruption) caused by lipid peroxidation alters phospholipid bilayer fluidity and protein positioning; transport proteins and enzymes embedded in the membrane lose functional orientation, reducing selective permeability and impairing substrate entry (e.g., glucose, oxygen), further limiting respiration and enzyme activity.

Low ATP levels reduce actin-myosin cross-bridge cycling in muscle fibres; ATP is required for detachment and re-cocking of myosin heads, therefore reduced ATP leads to weak muscle contraction, fatigue, and slow tissue repair.

(b) Strategies

- *Balanced diet provision supplies glucose, amino acids, and lipids as substrates for respiration, restoring enzyme activity and ATP production.*
- *Use of protective equipment prevents inhalation of inhibitors, maintaining enzyme structure and function.*
- *Improved ventilation systems reduces accumulation of toxic chemicals, preserving mitochondrial integrity.*
- *Proper industrial waste management, reduces environmental toxin exposure affecting cellular respiration.*
- *Regular health monitoring, detects early enzyme dysfunction and mitochondrial damage.*

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SYLLABUS ALIGNED NOTES

TOPICAL ASSESSEMNT ITEMS

END OF CYCLE TEST PAPERS

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