Data Response Practice (Ans)

PRACTICE 1

Pyruvate dehydrogenase is a tetrameric enzyme found in the mitochondria that catalyses the conversion of pyruvate to acetyl CoA during cellular respiration.

The activity of pyruvate dehydrogenase is inhibited by acetyl Co-A.

Fig. 1 shows the relationship between the initial rate of reaction of pyruvate dehydrogenase activity and the concentration of pyruvate at the optimal temperature and pH of the enzyme. All other variables are kept constant.

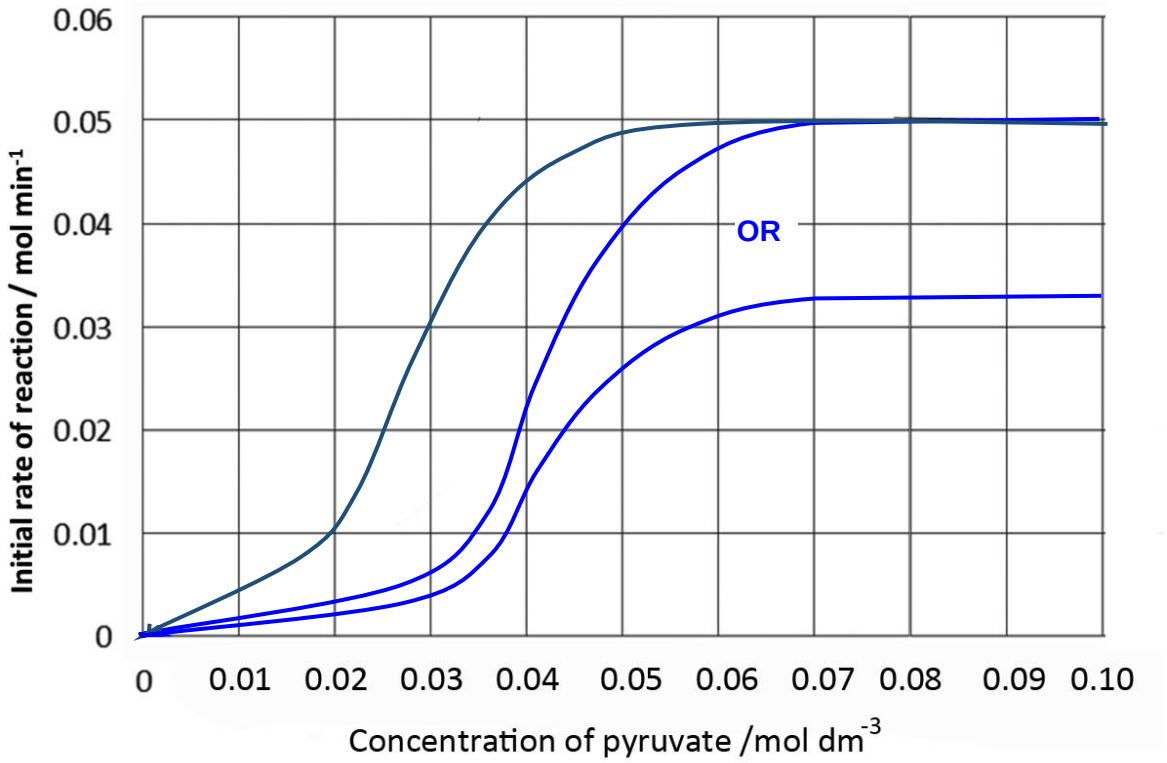

Fig 1

(a) Comment on the trend shown on the graph. **[HI2]** [6]

Describe + Explain

- [Describe] As the concentration of pyruvate increases from **0 to 0.02 moldm^{-3},** the **rate of reaction increases gradually from 0 molmin^{-1} to 0.01 molmin^{-1}.**

- *[Explain] At low pyruvate concentrations, the **inactive** form of the pyruvate dehydrogenase is favoured. / Only a **small number of enzymes** are able to form **enzyme-substrate complexes** per unit time.*

- [Describe] As the concentration of pyruvate **increases from 0.02 moldm^{-3} to 0.04 moldm^{-3},** the rate of reaction **increases steeply from 0.01 molmin^{-1} to (0.044 - 0.045) molmin^{-1}** (sigmoidal curve)

- *[Explain]* The substrate binds to **one active site** in the enzyme and causes a **conformational change** in the enzyme, hence, **stabilising the other subunits** in the **active form** and **increases the affinity** for binding to its substrate molecules.

- [Describe] As the concentration of pyruvate **increases from (0.057-0.059) moldm^{-3} to 0.10 moldm^{-3},** the rate of reaction **plateaus/remains constant at 0.05 molmin^{-1}.**

- [Explain] ***All active sites of all** pyruvate dehydrogenase molecules are **saturated** with **pyruvate molecules**.*

(b) On Fig 1, sketch and label a curve to show the effect of adding acetyl Co-A on the initial rate of reaction of pyruvate dehydrogenase activity. **[HI 1]**[1]

- Graph should show a lower initial rate of reaction over the entire range of pyruvate concentrations. Graph may reach the V_{max} of the normal graph or not reach it.

PRACTICE 2

Fig. 2 shows the effect of increasing temperature on the activity of three protein-digesting enzymes:

- thermitase from thermophilic *Thermoactinomyces vulgaris*
- subtilisin from *Bacillus subtilis*
- modified subtilisin

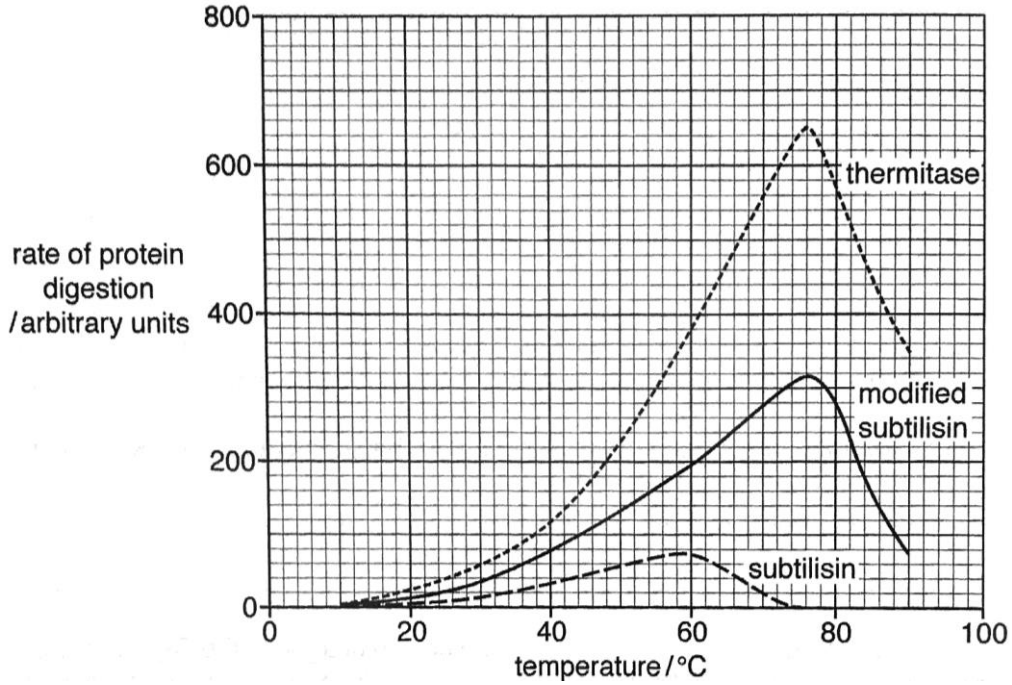

Fig. 2

Describe means "describe trend of graph and cite data only! No need to explain why!!!

(a) Describe, with reference to Fig. 2, the effect of temperature on the rate of protein digestion by thermitase. **[HI-1]** [3]

- As temperature increases from **10°C to 40°C**, rate of protein digestion **increases gradually** from **0 a.u. to 120 a.u**.
- As temperature increases from **40°C to 76°C**, the rate **increases steeply** from **120 a.u. to 650 a.u**.
- At **optimum temperature of 76°C,** rate of protein digestion is at its **maximum.**
- As temperature increases from **76°C to 90°C**, the rate **decreases steeply** from **650 a.u. to 350 a.u**.

(b) Modified subtilisin is similar to subtilisin but had eight of its amino acids replaced with different amino acids.

Describe **and** explain the effect of this modification on the activity of subtilisin. **[HI-2]** [4]

> **Important**: ***CONTEXTUALISE*** your answers. DO NOT give vague answers by mere description of how primary structure is changed and how it subsequently changes the secondary and tertiary structure of protein.

- **[Describe]** Rate of protein digestion is **higher with modified subtilisin** at every temperature investigated as compared to subtilisin, [Cite data – at any temperature] At **30°C**, the rate of reaction of subtilisin is **10 a.u**. whereas rate of modified substilisin is **40 a.u**.
- **[Explain]** The replaced amino acids might be the catalytic amino acids. The new amino acids may have **higher catalytic ability**
- **[Describe]** Modified subtilisin has **higher optimal temperature of 76°C** as compared to subtilisin with **optimal temperature of 58°C**
- **[Explain]** The new amino acids might be **cysteine** that forms **strong disulfide (covalent) bonds** that hold the **3D structure** of the enzyme **intact at higher temperature/ thermostable enzyme**.

PRACTICE 3

In 1954, an article was published in the British Medical Journal entitled, *The mortality of doctors in relation to their smoking habits*.

One aspect of the investigation studied a very large number of doctors in the UK aged 35 years and older. A survey established the quantity of tobacco smoked per day.

Twenty-nine months later, the case of any deaths in the study group was recorded.

Table 3 summarises the results obtained.

Table 3

Cause of death	Number of deaths	Death rate per year per 1000 men in the study			
		Non-smokers	Smokers, tobacco smoked / g day^{-1}		
			1-14	15-24	25 and above
Coronary thrombosis (heart attack)	235	3.89	3.91	4.71	5.15
Lung cancer	36	0.00	0.48	0.67	1.14

(a) State which group in the study is most at risk from dying of lung cancer. **[HI-1]** [1]

- Smokers who smoked **25g day^{-1} and more** tobacco.
  (idea of tobacco smoked with units must be clear)

Examiner's comments

- A handful of candidates were not specific and identified 'smokers' without specifying the different groups.
- Some candidates lost marks because units were either not given or wrongly identified.

(b) Using information from Table 3 to support your answer, discuss the evidence linking tobacco smoking to coronary thrombosis and early death. **[HI-3]** [4]

- Death rate caused by coronary thrombosis is higher in smokers than non-smokers.
- **[cite relevant data]** Smokers who smoked 1-14 g day^{-1} has a death rate of 3.91 per year per 1000 men which is higher than non-smokers which have a death rate of 3.89.
- As the amount of tobacco smoked increased, the death rate per year per 1000 men caused by coronary thrombosis increases.
- **[cite data]** As the amount of tobacco smoked increased from 1-14 g day^{-1} to 25 g day^{-1} and above, the death rate per year per 1000 men increased from 3.91 to 5.15.

Examiner's comments

- Candidates did not read the question carefully and end up discuss about lung cancer when it is not needed.
- Weaker students failed to cite data despite the phrase 'Using information from Table3.1'.
- Most only gave 1 conclusion of trend seen.

(c) Suggest a significant limitation of this study. **[KU-3]** [1]

- No females are included in the study.
- Deaths as a proportion of the sample is lacking.
- Information (e.g. age group, family history) are not considered.
- Other diseases and deaths caused by smoking tobacco are not included.
- AVP

Examiner's comments

- Sample size of 1000is a substantial number.
- Period of investigation too short was not accepted.

PRACTICE 4

p53 is a tumor suppressor protein which plays an important role at cell cycle checkpoints. An experiment was carried out to investigate the effect of p53 on the cell cycle in human liver cells. Two types of cells were used in the investigation, one with normal *p53* gene and the other with mutated *p53* gene, resulting in p53-deficient cells. Both cell types were subjected to γ-radiation, which is a DNA damaging agent.

Mitotic index of the cells were then measured and the results are shown in Fig. 4. Mitotic index reflects the percentage of cells in a population that are dividing. It is calculated by counting the number of cells with condensed chromosomes and dividing it by the total number of cells observed.

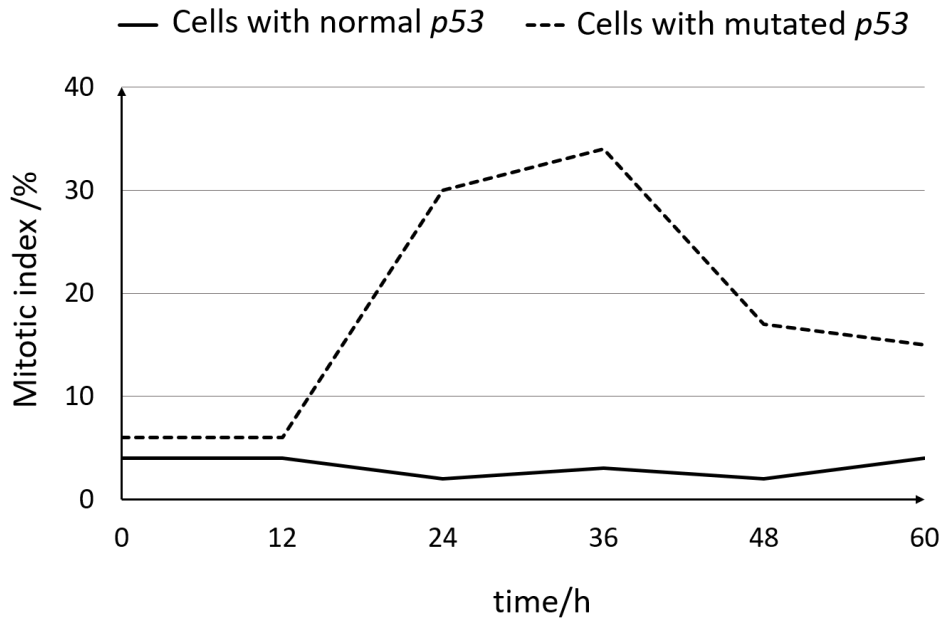

Fig. 4

a) **[HI-2]** With reference to Fig. 4, account for the difference in results obtained after 12 hours. [3]

[Describe]

- [trend] Cells with mutated *p53* gene has a **higher** mitotic index **at all times/from 12h-60h** compared to cells with normal *p53* gene.

- [cite data] the mitotic index for cells with normal *p53* gene is always **below 5%** whereas the mitotic index for cells with mutated *p53* gene **increased steeply from 12 hours to 24 hours, from 6% to 30%** (or data till 36 hours).

[Explain]

- Normal p53 protein **halts the cell cycle** and hence results in lower mitotic index in the presence of DNA damage, whereas p53-deficient cells will continue **progressing on to mitosis** resulting in higher mitotic index

PRACTICE 5

A chemical substance, 2-carboxy-D-arabitinol 1-phosphate (also known as CA1P), is naturally found in plants and it is structurally similar (analogue) to ribulose bisphosphate (RuBP).

Fig. 5 is a graph showing the effects of adding CA1P to a sample of purified chloroplasts. A control containing chloroplasts in the absence of CA1P was also set up. Light intensity, carbon dioxide concentration and temperature were kept constant.

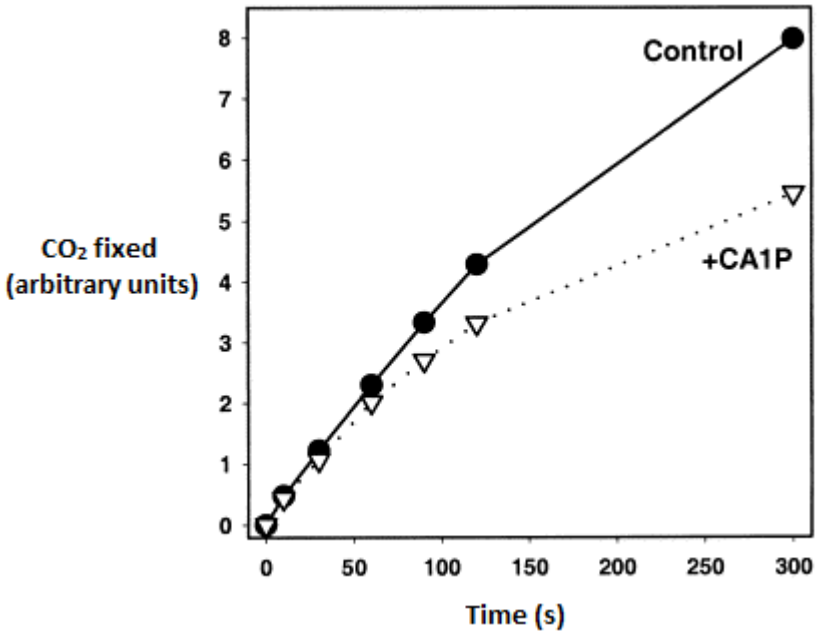

Fig. 5

(a) With reference to Fig. 5, describe and explain the effect of CA1P on carbon fixation. [4]

[Compulsory] Describe:

- **When CA1P is present**, the **amount of CO_2 fixed at 300s** was **lower at 5.5 a.u. (accept 5.3-5.5)** **compared with the control** where the **amount of CO_2 fixed at 300s is 8.0 a.u. (accept 7.8-8.0).**

Explain:

- CA1P is a **competitive inhibitor** of **Rubisco**
- CA1P is **structurally similar to RuBP** hence **competes with RuBP for the active site of RuBP carboxylase**
- Leading to a **decrease in the rate of enzyme-substrate complexes** formed.

PRACTICE 6

A recent research study indicates that bed bugs have increased resistance to neonicotinoids, the most widely used insecticide in the world. Resistant strains were found to have elevated detoxifying enzymes. Scientists say non-chemical methods of control now need to be considered.

Fig. 6 shows the volumetric units of insecticides used per thousand household per month, over a period of eleven years from 1984 to 1994. The figure also shows the percentage of infestations each year caused by resistant strains of *Cimex lectularius*.

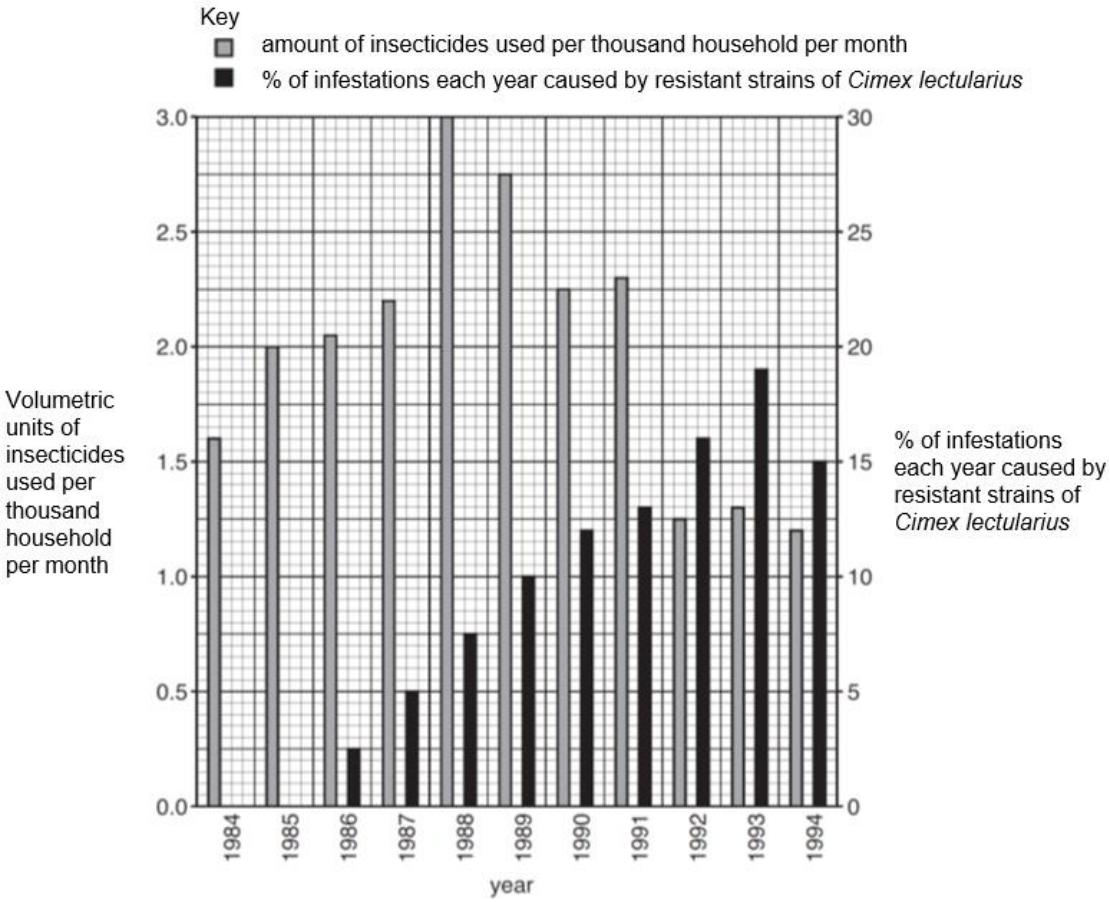

Fig. 6

(a) With reference to Fig. 6,

(i) Describe and explain the trend in percentage of infestations each year caused by resistant strains of *Cimex lectularius* between 1986 and 1993. **[HI-2]** [3]

- **[Compulsory]** percentage of infestations **increased** from **2.5%** in 1986 to **19%** in 1993.
- **Use of insecticides** act as a **selection pressure**.
- **Insecticide resistant bedbugs** have a **selective advantage**, so they can **survive, reproduce and pass on their advantegous alleles** to fertile and viable offspring.
- Hence **increasing the frequency of insecticide resistant alleles** and hence more resistant strains of *Cimex lectularius* in the bed bug population.

(ii) Use evidence from the graph to suggest why the percentage of infestations each year caused by resistant strains of *Cimex lectularius* decreased in 1994 compared to previous years. **[HI-2]** [2]

- **[Compulsory] Lower amount of insecticides used per thousand households per month** used between **1992 to 1994 (1.25, 1.3 and 1.2 volumetric units)**
- **…therefore more non-resistant strains can survive** and **reproduce** to increase the population size
- hence, **proportion** of the resistant strains **decreases**.

PRACTICE 7

People with Alzheimer's disease (AD) lose their ability to form new memories. One form of AD is caused by a mutation of the amyloid precursor protein (APP) gene, which encodes the APP transmembrane glycoprotein. when APP is incorrectly cleaved, an insoluble protein known as β-amyloid will be formed. This results in the formation of hard, insoluble plaques. These plaques accumulate in the brain of an individual with AD.

It was hypothesised that plaque formation was dependent on two critical factors:

- the type of organism – human and mouse
- the type of cells – nerve and monocytes.

To investigate these factors, a solution containing a fixed concentration of β-amyloid was added to three different samples labelled **1** to **3** and incubated for a fixed duration of time.

Fig. 7.1 shows plaque formation after the incubation, as measured and recorded as percentage plaque formation.

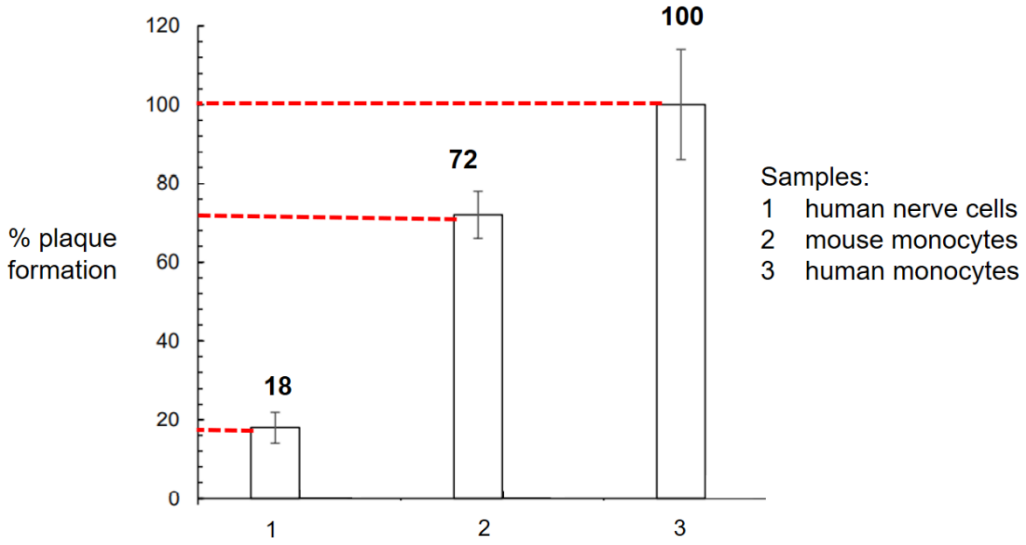

Fig. 7.1

(a) With reference to Fig. 7.1, evaluate the extent to which these factors promote plaque formation. **[HI-3]** [4]

Type of organism

- **Human monocytes** promote plaque formation to a **greater extent** than **mouse monocytes**.

OR

- Both **human and mouse monocytes** have a **high percentage of plaque formation** despite being different organism.
- **[Compulsory] There is 72% plaque formation in mouse monocytes** while 1**00% plaque formation in human monocytes**.

Type of cells

- **Monocytes** promote plaque formation to a **greater extent** than **nerve cells**
- **[Compulsory]** There is 1**00% plaque formation in monocytes** which is **higher** than in **nerve cells** with **18% plaque formation.**

Compare between type of cell and type of organism

- **Type of cell has a greater extent** in promoting plaque formation **than type of organism**.
- The **difference in % of plaque formation between human monocyte and human nerve cells** is **82%** [1] which is **greater** than **difference in % of plaque formation between human monocyte and mouse monocyte which is 28%** [1]

Further investigation was conducted to find out the effect of heating on plaque formation in human monocytes. A solution containing a fixed concentration of β-amyloid was added to a sample of heat-killed monocytes and living monocytes respectively. These samples were then incubated for a fixed duration of time. Control for each sample was set up where no β-amyloid was added.

Fig. 7.2 shows the results of the investigation.

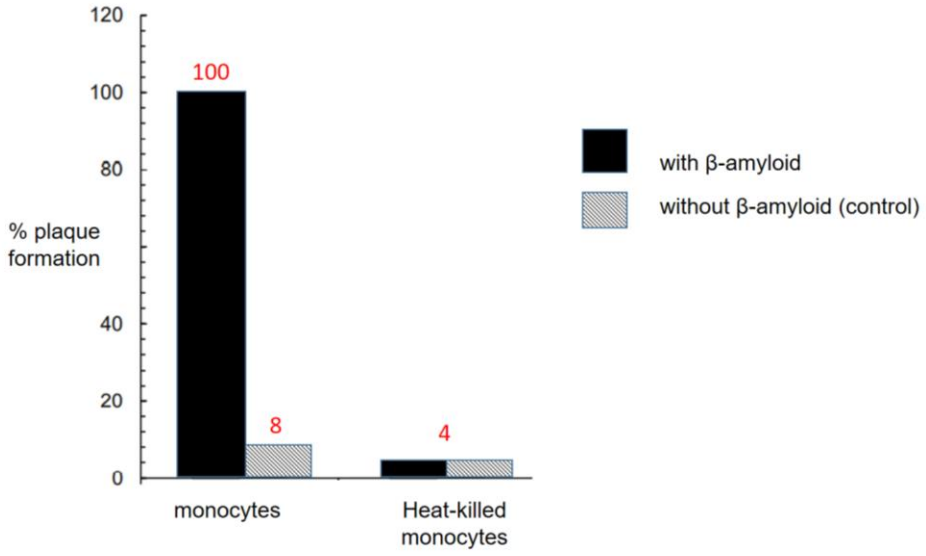

Fig. 7.2

(b) With reference to Fig. 7.2, describe the **effect of heating** on plaque formation in monocytes with and without β-amyloid. **[HI-2]** [2]

- With β-amyloid, human monocytes showed a **larger decrease** in % of plaque formation compared to human monocytes without β-amyloid.

- **With β-amyloid**, % of plaque formation **decreased from 100% to 4%** (by 12.5 times) **when heat-killed**, while monocytes **without β-amyloid decreased from 8% to 4%**. [Accept: 5%].

(c) Another genetic disease that leads to a loss of brain function is Huntington's disease. The gene involved contains a section of DNA with many repeats of the base sequence CAG. The number of these repeats determines whether or not an allele of this gene will cause Huntington's disease.

This disease allele is dominant

- An allele with 40 or more CAG repeats will cause Huntington's disease.
- An allele with 36-39 CAG repeats may cause Huntington's disease.
- An allele with fewer than 36 CAG repeats will not cause Huntington's disease.

Fig. 7.3 shows the age at which a sample of patients with Huntington's disease first developed symptoms and the number of CAG repeats in the allele causing Huntington's disease in each patient.

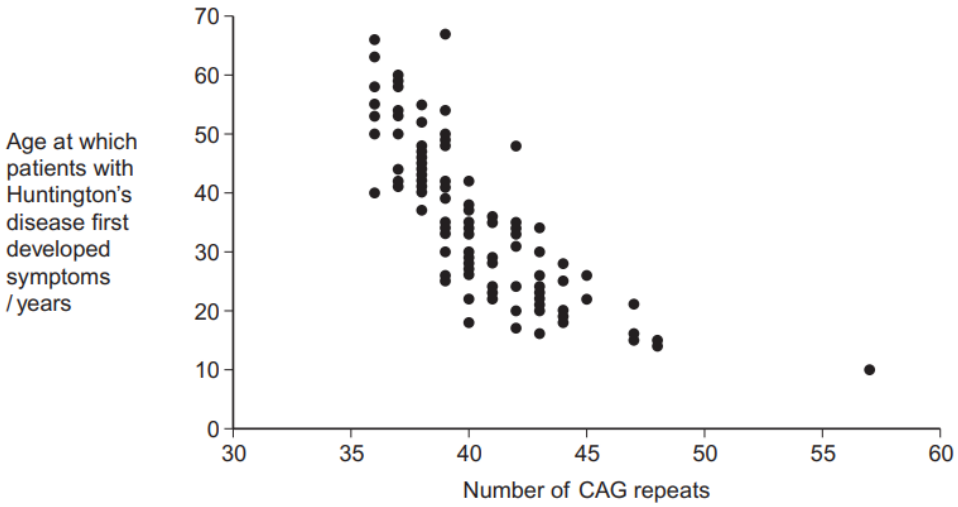

Fig. 7.3

(i) People can be tested to see whether they have an allele for this gene with more than 36 CAG repeats. Some doctors suggest that the results can be used to predict the age at which someone will develop Huntington's disease.

Use the information in Fig. 7.3 to evaluate this suggestion. **[HI-2]** [2]

- **Negative correlation** between CAG repeats and age when symptoms first developed / As no. of CAG repeats increases, the earlier the age when symptoms first developed.

- There is a **wide range of age of onset** with a specific no. of CAG repeats

- e.g. individuals with CAG repeats of 40, the age when symptoms first developed ranges from 18 to 42

(ii) Huntington's disease is always fatal. Despite this, the allele is passed on in human populations.

With reference to Fig. 7.3, suggest and explain why this is so. **[HI-2]** [2]

- **[Suggest]** The earliest age on onset is 36 years old…
- **[Explain]** …so these individuals may **already have had children and passed on the alleles to them**.

PRACTICE 8

Unlike the animals where the oxygen is obtained through breathing, plants have to first synthesize oxygen through photosynthesis.

A scientist carried out the following experiment to investigate the effect of light intensity on the rate of oxygen produced from a water plant, *Elodea*.

- *Elodea* was cut into three pieces, each 10 cm long.
- Each piece of *Elodea* was placed in a glass tube, containing 0.5% sodium hydrogen carbonate solution, which was then sealed with a bung.
- Tube **A** was placed 10 cm away from a lamp.
- Tube **B** was placed 5 cm away from a lamp.
- Tube **C** was placed in a dark room.
- An oxygen sensor was used to measure the percentage of oxygen in the solutions at the start of the experiment and again at 5, 10 and 20 minutes.

The results are shown in **Fig. 8**.

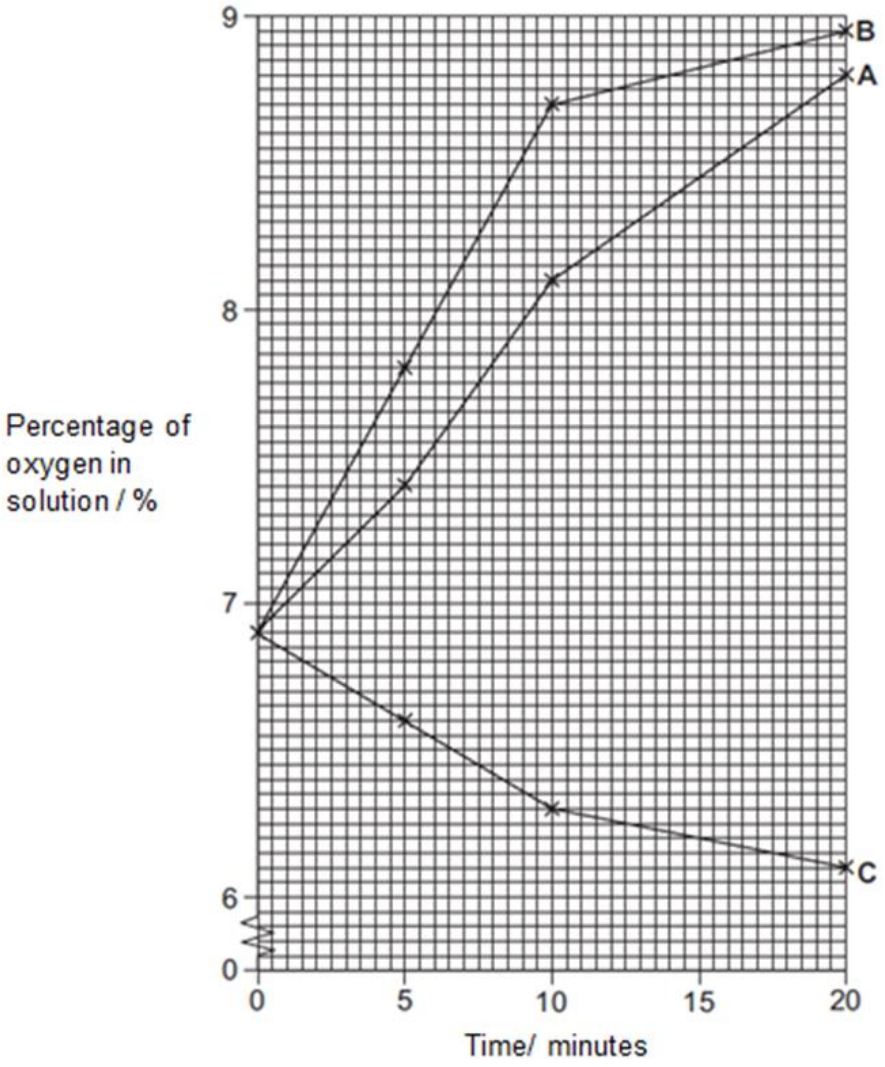

Fig. 8

(a) With reference to **Fig. 8**, calculate the rate of oxygen production for tube **A** from 0 to 5 minutes of the experiment.

Express your answer to two decimal places. Show your working. **[HI-2]** [1]

Calculation

1. (7.4% - 6.9%) / 5 = **0.10 % per minute**

(b) **Compare** the results for tubes **A** and **B**. **[HI-2]** [4]

[Similarity]

1. [Trend] Both tube A and B showed an **increase in the percentage O_2** produced **from 0-20min.**

2. [cite data] From **0-20min**, percentage O_2 in solution **B** increased from **6.9% to 8.95%** but percentage O_2 in solution A increased from **6.9% to 8.8%**

[Difference]

3. [Trend] Percentage O_2 produced in tube **B** is **higher** than the percentage O_2 produced in tube **A** **from 0 to 20 min**.
4. [Cite any data]
   E.g. At 5 min, percentage O_2 produced in tube **B** is **7.8%** but percentage O_2 in solution A is **7.4%**.

OR

5. [Trend] From **0-10min**, percentage O_2 in tube **B increased more steeply** than percentage O_2 produced in tube **A**

6. [Cite data] From **0-10min**, percentage of O_2 in tube **B has a higher increase of 1.8%** but O_2 increase in solution **A increased by only 1.2%**.

OR

7. [Trend] From **10-20min**, percentage O_2 in tube **B increased less steeply** compared to percentage O_2 produced in tube A

8. [Cite data] From **10-20min**, percentage of O_2 in tube **B has a lower increase of 0.25%** but O_2 in tube **A** has a **higher increase of 0.7%**.

(c) Account for the result for tube **C**. **[HI-2]** [2]

Transformation of energy

9. [cite data] From **0 – 20 min**, percentage of O_2 in solution C decreased from **6.9% to 6.1%**

10. [explain] In the **absence of light**, **no oxygen** is produced **AND oxygen is used in aerobic respiration**

PRACTICE 9

In an attempt to control the spread of dengue, using genetic modification, a piece of DNA is inserted into the *Aedes aegypti* mosquito genome at the embryonic stage. This DNA contains a lethal gene (*tTAV* gene) which codes for a protein called tTAV. This protein acts as a molecular switch to shut down the expression of **all** other genes, leading to death of the insect.

This tTAV protein, however, is inactivated by a compound called tetracycline, which is incorporated into the food that the developing larvae feed on. Hence, the genetically-modified (GM) larvae survive to adulthood, with much of the tetracycline still remaining in them. Male GM mosquitoes are then selected to breed with females to produce large number of offspring. The male GM offspring are selected and fed with tetracycline until they reach adulthood. They are then released into the wild to mate with wild-type females. Any offspring larvae produced will contain the *tTAV* gene, which is expressed to cause death of the larvae.

(a) **[HI-2]** Suggest **two** advantages of using GM mosquitoes over the use of pesticides in controlling the spread of dengue. [2]

- ***Idea that*** Highly species-specific: the released male GM mosquitoes only mate with the females of their own species. This means that no other insects are affected, unlike the use of pesticides that affects all insects

- ***Idea that*** Male GM mosquitoes does not pose any harmful environmental effects, unlike the chemicals found in pesticides.

- ***Idea that*** Mosquitoes can develop resistance against pesticide

Male GM mosquitoes have been used in open field trials in countries such as Cayman Islands. The town where the *Aedes aegypti* mosquitoes predominate was divided into three areas, as shown in Fig. 9.1.

Area **A** – the treatment site where male GM mosquitoes are released
Area **B** – buffer zone
Area **C** – the non-treated control site

The mosquito populations in area **A** and area **C** were measured using an ovitrap – a device that is attractive as an egg-laying site for female mosquitoes.

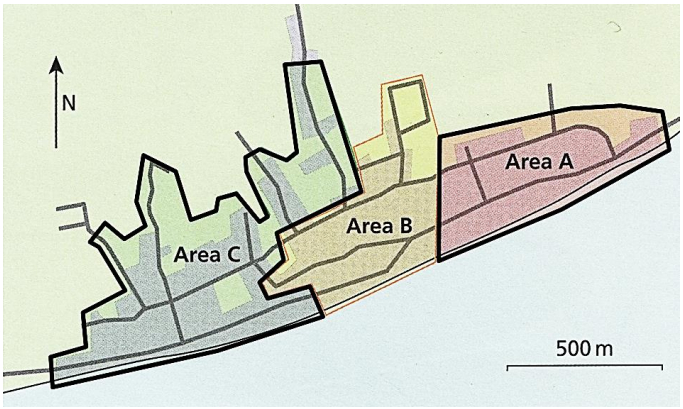

Fig. 9.1

(b) **(i)** State why the release of male GM mosquitoes in area **A** will not increase the risk of transmission of dengue. [1]

- ***Idea that*** Only female mosquitoes feed on human blood to transmit the dengue virus.

(ii) Suggest the purpose of area **B**. [1]

- ***Idea that*** Ensure GM mosquitoes were unlikely to fly to non-treated control site, which could interfere with the results.

The number of ovitraps that contain eggs were recorded every week for 6 months. Fig. 9.2 shows the ovitrap index, which is calculated based on the percentage of ovitraps containing eggs.

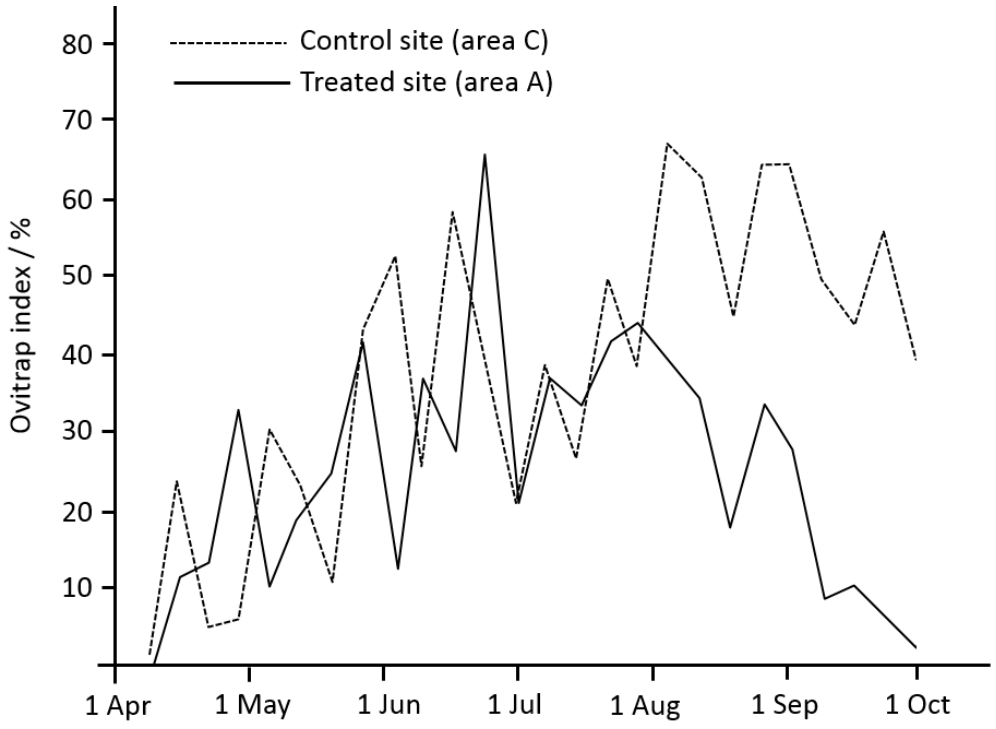

Fig. 9.2

(c) [HI-3] **Comment on** the trend observed for both the treated and control site. [4]

- For both sites, ovitrap index **fluctuates** from **Apr to Oct**.
- **Rises** due to **females laying eggs**, **falls** due to the **hatching of the eggs**
- For **control site**, **upward trend from Apr to Oct**, but for **treated site**, **upward trend from Apr to mid-Jun to Aug**, then **downward trend till Oct**.
- By **1 Oct**, ovitrap index stands at **40% at control site**, but only **2% at treated site**
- Initial increase from Apr to Oct in treated site as there are still non-GM mosquitoes to mate and produce viable offspring.
- However, in non-treated site, male GM mosquitoes pass on the tTAV gene to offspring, causing the **larvae** to **die** before reaching adulthood, hence less adult mosquitoes mate to lay eggs.

End of Paper