RIVER VALLEY HIGH SCHOOL
YEAR 6
PRELIMINARY EXAMINATION II

CANDIDATE NAME											
CENTRE NUMBER	S					CLASS		INDEX NUMBER				

H2 BIOLOGY **9744/02**

Paper 2 Structured Questions **11 Sep 2017**

2 hours

Candidates answer on the Question Paper.

No Additional Materials are required.

READ THESE INSTRUCTIONS FIRST

Write your Centre number, index number and name in the spaces at the top of this page.
Write in dark blue or black pen.
You may use an HB pencil for any diagrams or graphs.
Do not use staples, paper clips, glue or correction fluid.
DO **NOT** WRITE IN ANY BARCODES.

Answer **all** questions in the spaces provided on the Question Paper.

The use of an approved scientific calculator is expected, where appropriate.
You may lose marks if you do not show your working or if you do not use appropriate units.

The number of marks is given in brackets [ ] at the end of each question or part question.

For Examiner's Use	
1	/ 9
2	/ 13
3	/ 8
4	/ 10
5	/ 12
6	/ 15
7	/ 10
8	/ 11
9	/ 12
Total	/ 100

This document consists of **24** printed pages.

Answer **all** questions.

1 Fig. 1.1 shows an electron micrograph of part of a plant cell.

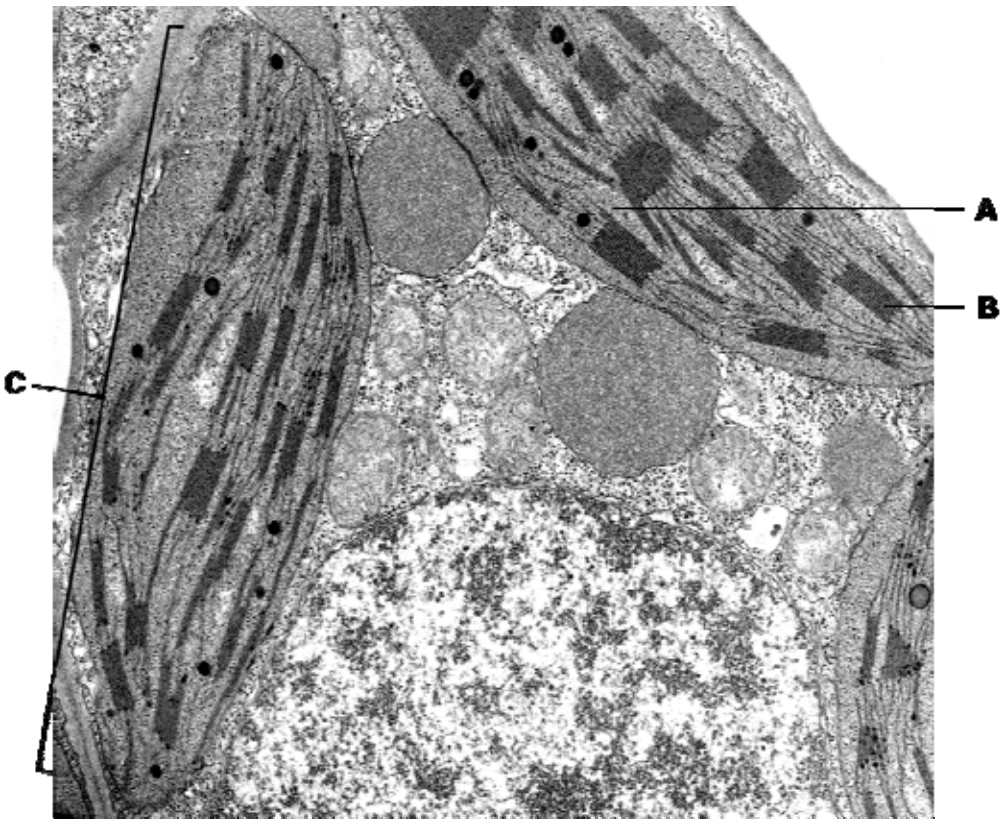


Fig. 1.1

(a) Identify region **A** and state its function. [2]

(b) Describe how the structure of the membrane at **B** allows it to perform its function. [3]

Cyanobacteria are prokaryotic cells that are capable of carrying out photosynthesis. The structure of a cyanobacteria is shown in Fig. 1.2.

Fig. 1.2

(c) With reference to Fig. 1.1 and Fig. 1.2, compare the visible structures of cyanobacteria with that of **C**. [2]

Cyanobacteria are considered to be the ancestors of structure **C**. They continued to function after being engulfed by primitive eukaryotic cells and evolved over time. This theory is known as the endosymbiont hypothesis.

(d) State **two** features of structure **C** that provide support for this hypothesis. [2]

[Total: 9]

2 Fig. 2.1 shows the structure of a G-protein coupled receptor (GPCR).

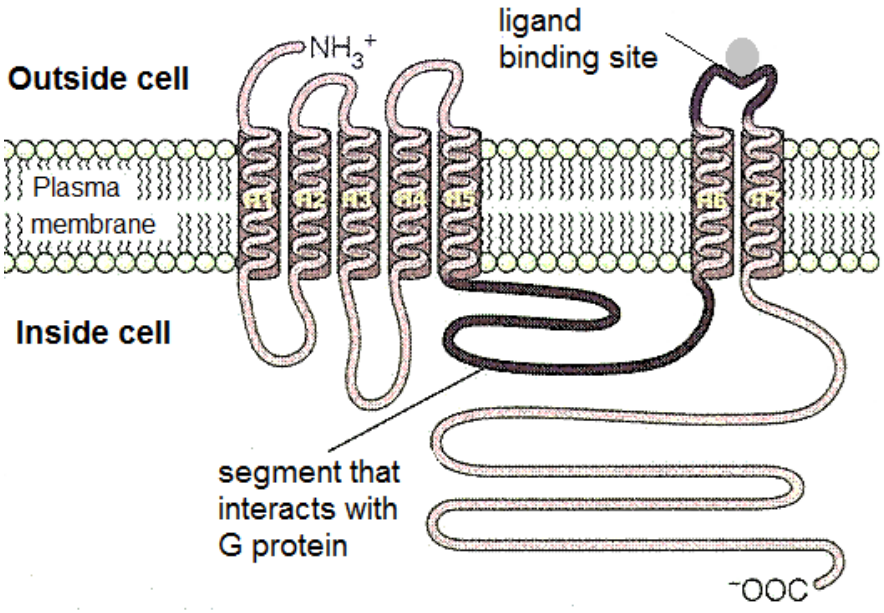


Fig. 2.1

(a) Describe how the structure of GPCR is adapted to its function. [3]

One of the cellular events resulting from glucagon binding to GPCR, shown in Fig. 2.1, is the activation of glycogen phosphorylase which breaks down glycogen to glucose.

(b) **(i)** Describe how binding of glucagon leads to activation of glycogen phosphorylase. [3]

(ii) Explain why liver cells store glucose in the form of glycogen. [3]

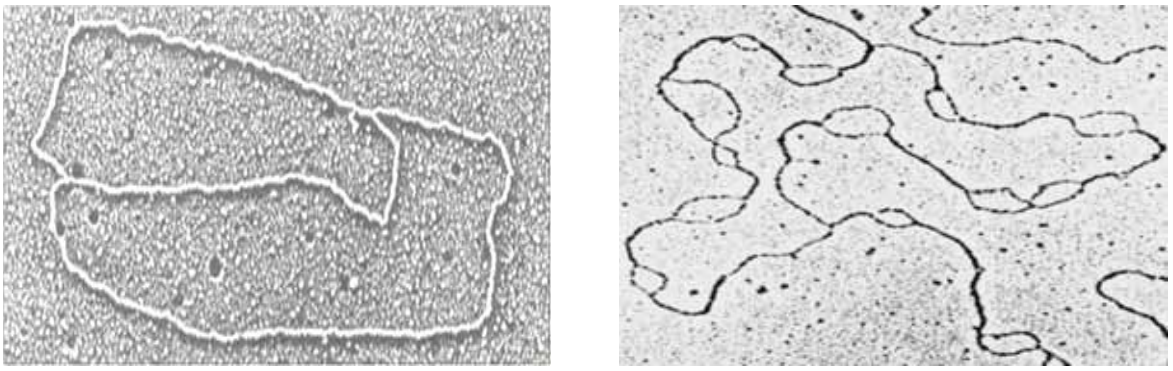
The binding of glucagon to GPCR leads to an increase in blood glucose level partly due to the action of glucose transporters. Glucose transporters transport glucose via facilitated diffusion.

(c) (i) Explain what is meant by facilitated diffusion. [2]

(ii) Explain why glucose transporters are necessary to facilitate this process. [2]

[Total: 13]

3 Fig. 3.1 shows DNA replication in an *Escherichia coli* (**A**) and in a mammalian cell (**B**). Diagrams are not shown to scale.



A **B**

Fig. 3.1

(a) State **one** way in which the DNA replication in these two organisms differs and explain the advantage of this to the mammalian cell. [2]

(b) Explain why DNA replication is said to be semi-conservative. [2]

End replication problem is a fundamental problem associated with replicating DNA in eukaryotes.

Some cells contain telomerase, which is responsible for extending the ends of DNA in eukaryotes. Fig. 3.2 shows the action of a telomerase enzyme.

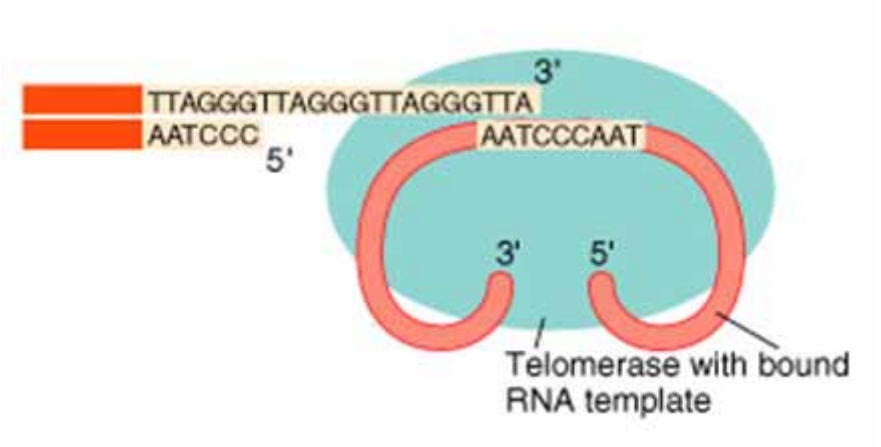


Fig. 3.2

(c) Explain how the end-replication problem arises. [2]

(d) With reference to Fig. 3.2, state **two** differences between transcription and the process of lengthening of DNA ends. [2]

[Total: 8]

4 Huntington's disease is a rare neurodegenerative disorder targeting the central nervous system. Transcriptional dysregulation is one of the commonly observed molecular abnormalities affected in this disease. Recent evidence suggests the involvement of a mutant Huntingtin protein in the processes regulating condensation of DNA, leading to activation of DNA damage response and death of nerve cells. DNA in various levels of condensation can be observed in the nerve cell nucleus. Fig. 4.1 shows one of the levels of condensation of chromatin.

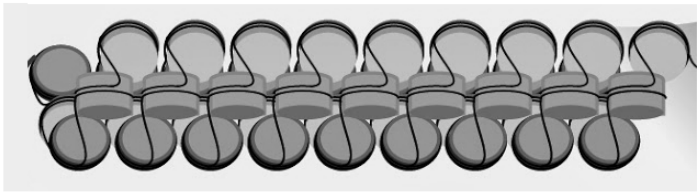


Fig. 4.1

(a) It is postulated that mutant Huntingtin protein facilitates packing of DNA into structure shown in Fig. 4.1. Describe how the DNA double helix is condensed into this structure. [2]

(b) The chromosomal condensation in **(a)** is the main reason for the commonly observed transcriptional dysregulation in Huntington's disease. Explain how transcription is affected. [3]

It is observed that nerve cells could remove Huntingtin proteins via ubiquitination of specific amino acids. However, the mechanism that triggers ubiquitination is unclear. In a study to determine the mechanism for degradation of Huntingtin proteins, selected amino acids were investigated and the results are shown in Table 4.1.

Table 4.1

	13th amino acid: serine	**16th amino acid: serine**	**6th amino acid: lysine**	**9th amino acid: lysine**	**15th amino acid: lysine**	**Fate of Huntingtin protein**
Trial 1	de-phosphorylated	de-phosphorylated	ubiquitin not attached	ubiquitin not attached	ubiquitin not attached	remains active
Trial 2	phosphorylated	de-phosphorylated	ubiquitin not attached	ubiquitin not attached	ubiquitin not attached	remains active
Trial 3	de-phosphorylated	phosphorylated	ubiquitin not attached	ubiquitin not attached	ubiquitin not attached	remains active
Trial 4	phosphorylated	phosphorylated	ubiquitin attached	ubiquitin attached	ubiquitin attached	degraded

(c) With reference to Table 4.1,

(i) state the level of control for Huntingtin gene expression. [1]

(ii) describe the events at the selected amino acids that triggers the degradation of Huntingtin proteins. [2]

(iii) describe how ubiquitination results in the removal of mutant Huntingtin protein. [2]

[Total: 10]

5 Fig. 5.1 shows the structure of a T4 virus.

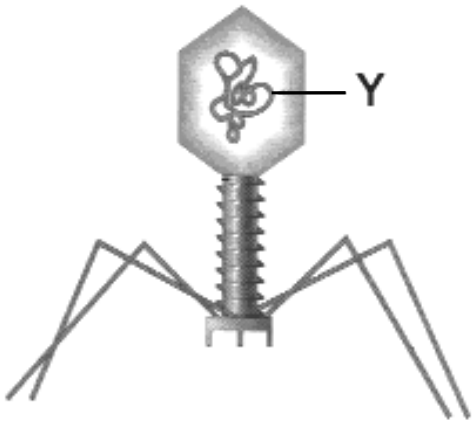


Fig. 5.1

(a) Identify structure **Y**. [1]

The T4 virus cannot reproduce by itself and relies upon a host cell for reproduction.

(b) State specifically why T4 viruses rely on host cells for their reproduction. [2]

T4 viruses use bacteria as its host. Fig. 5.2 shows the results of an experiment in which T4 viruses were added to a culture of bacteria. Samples of the culture were then taken at intervals to determine the number of free T4 viruses present.

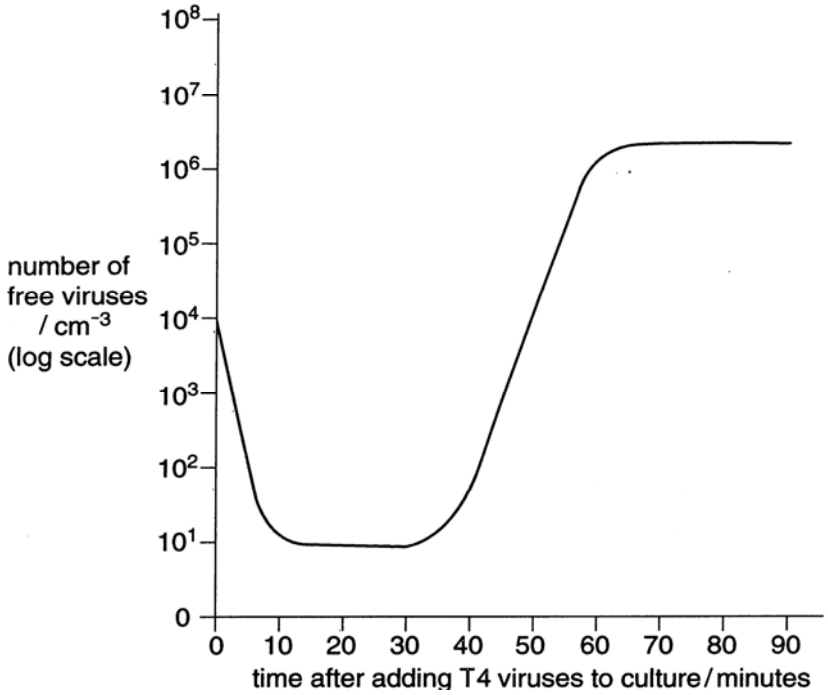


Fig. 5.2

(c) With reference to Fig. 5.2, describe and explain the changes in number of free T4 viruses

(i) in the first 10 minutes; [2]

(ii) between 30 and 60 minutes. [3]

A scientist carried out an investigation using T4 virus and two strains of bacteria: $\mathbf{B^+}$ cells which can grow in media without lysine and $\mathbf{B^-}$ cells which only grow when supplied with lysine. The procedure is shown in Fig. 5.3.

T4 are mixed with $\mathbf{B^+}$ cells

↓

T4 are isolated from the culture and added to $\mathbf{B^-}$ cells

↓

$\mathbf{B^-}$ cells are plated on medium lacking lysine

↓

Growth observed on medium

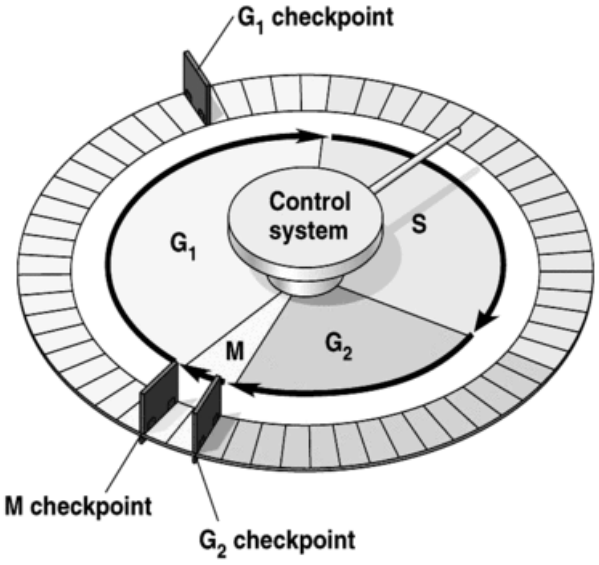
Fig. 5.3

(d) (i) Explain the observations made by the scientist. [3]

(ii) Suggest **one** other potential benefit of the process mentioned in **(d)(i)** for the recipient bacteria. [1]

[Total: 12]

6 The cell cycle is an ordered sequence of events involving two stages that culminates in cell growth and division into daughter cells. It is an essential mechanism by which all living things reproduce.



Pearson Education Inc., 2017

Fig. 6.1

(a) With reference to Fig. 6.1, name the longest stage of the cell cycle and discuss the main events in this stage. [3]

Fig. 6.2 shows a cell viewed from the spindle pole during cell cycle.

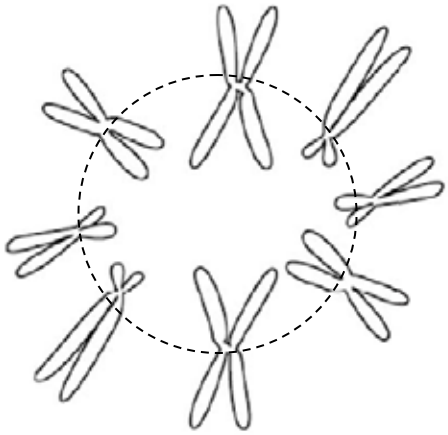


Fig. 6.2

(b) **(i)** State the type of nuclear division and name the stage shown in Fig. 6.2. [1]

type of nuclear division _____

stage _____

(ii) Explain your answer for **(b)(i)**. [2]

_____

_____

_____

_____

(c) With reference to Fig. 6.2, complete Table 6.1 to show the number of chromosomes and mass of DNA in each nucleus during different phases of mitosis. [2½]

Table 6.1

	Number of chromosomes per nucleus	**Mass of DNA per nucleus / µg**
Prophase of mitosis		170
Metaphase of mitosis		
Telophase of mitosis		

Mutations in *ras* proto-oncogenes are among the most common events in cancer. Gain-of-function mutations in *ras* proto-oncogenes are known to result in dysregulation of the cell cycle due to faults in signalling pathways.

(d) Explain what is meant by proto-oncogenes. [1½]

(e) Explain how a mutant Ras protein may lead to cancer. [3]

(f) Other than cancer cells, *ras* gene expression is also upregulated in embryonic stem cells. However, the latter does not result in a disease phenotype.

Explain what embryonic stem cells are. [2]

[Total: 15]

7 The coat colour of Labrador retriever dogs are determined by genes at two loci. The presence of the dominant alleles **B** and **E** results in black coats, whilst the presence of only the dominant allele **E** results in brown coats. Individuals that are homozygous recessive at the **E/e** locus will have golden coats.

A true breeding male retriever with a black coat was crossed with a female retriever with a golden coat. The resulting F_1 offspring all had black coats and the same genotype. A test cross was conducted for the F_1 individuals.

(a) State the genotype of the F_1 individuals. [1]

(b) Using the symbols for the alleles stated above, draw a genetic diagram to explain the test cross. [3]

(c) Name and describe the type of interaction between the gene loci. [3]

The pedigree shown in Fig. 7.1 shows the inheritance of coat colour in a family of Labrador retrievers.

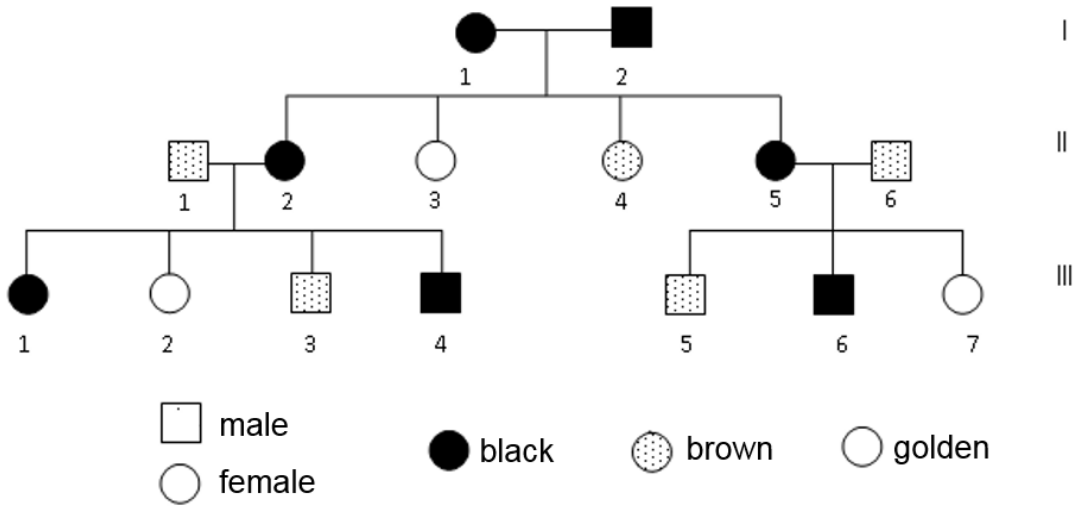


Fig. 7.1

(d) **(i)** State the genotype of individual II-1. [1]

(ii) Explain your answer in **d(i)**. [2]

[Total: 10]

8 Fig. 8.1 is an electron micrograph of a mitochondrion.

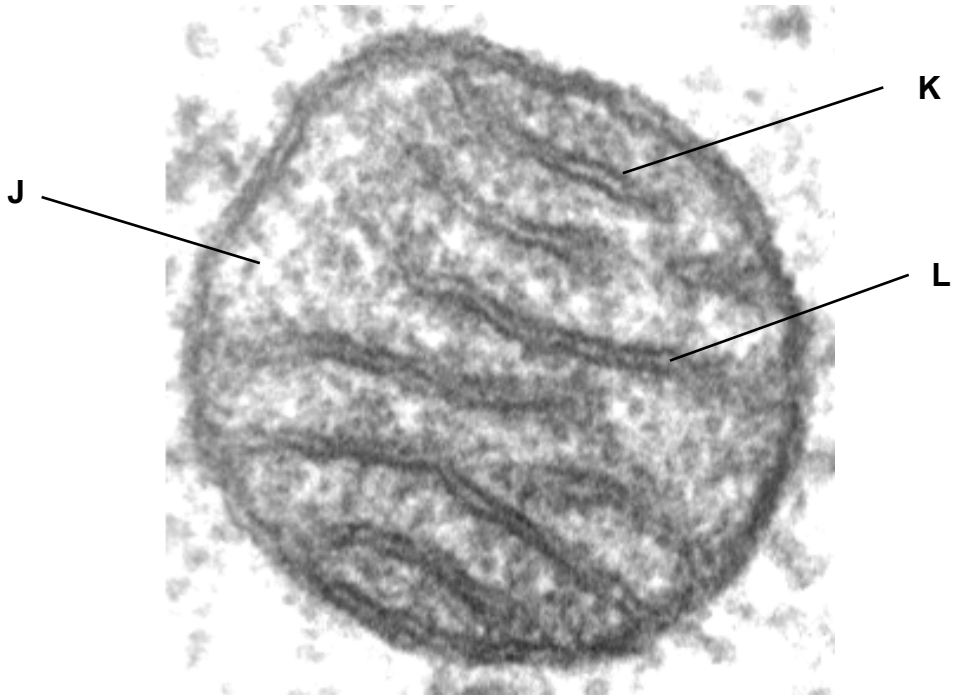


Fig. 8.1

(a) (i) Identify structures **J** and **K**. [1]

J _____

K _____

(ii) Describe how structure **J** is adapted to its function. [1]

_____

_____

(b) (i) State the role of high concentration of protons at **L**. [1]

_____

_____

(ii) Explain how the high concentration of protons is generated at **L**. [3]

In an investigation to determine the effect of chemical **M** on respiration, mitochondria were incubated in four ways:

1. with glucose
2. with pyruvate
3. with glucose and chemical **M**
4. with pyruvate and chemical **M**

The results are summarised in Table 8.1.

Table 8.1

	CO_2 evolution	O_2 consumption	ATP production by oxidative phosphorylation
Glucose	x	x	x
Pyruvate	✓	✓	✓
Glucose + chemical **M**	x	x	x
Pyruvate + chemical **M**	✓	✓	x

(c) (i) Explain why carbon dioxide is produced when mitochondria are incubated with pyruvate but not when incubated with glucose. [3]

(ii) Suggest why when mitochondria were incubated with pyruvate and chemical **M**, oxygen consumption occurs but not ATP production. [2]

[Total: 11]

9 Tetanus is a disease caused by a bacterium. When the tetanus bacteria enter the body they release a toxin which causes muscular rigidity and extreme pain. Children in the United Kingdom are routinely vaccinated against tetanus at an early age.

Fig. 9.1 is a diagram that shows three B lymphocytes (**P**, **Q** and **R**) and the events that occur during an immune response to the tetanus toxin.

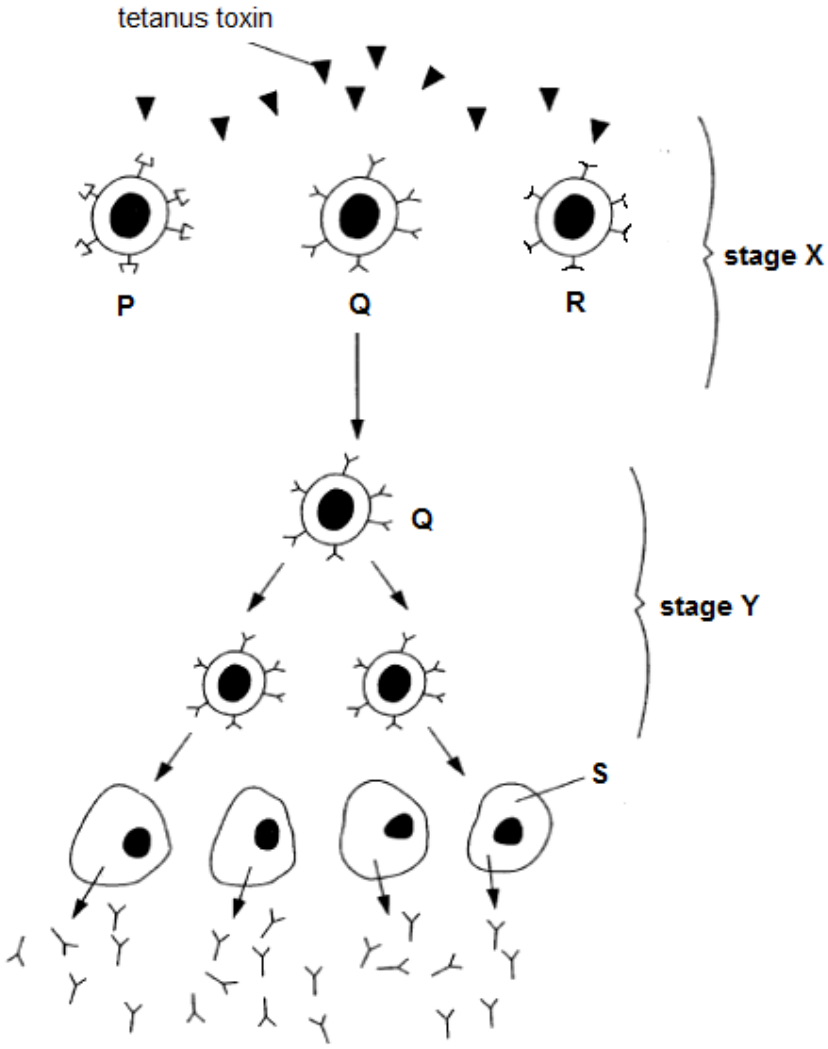


Fig. 9.1

(a) Explain what is happening at stages **X** and **Y** in the immune response to tetanus toxin. [2]

Fig. 9.2 shows an antibody molecule secreted by cell **S**.

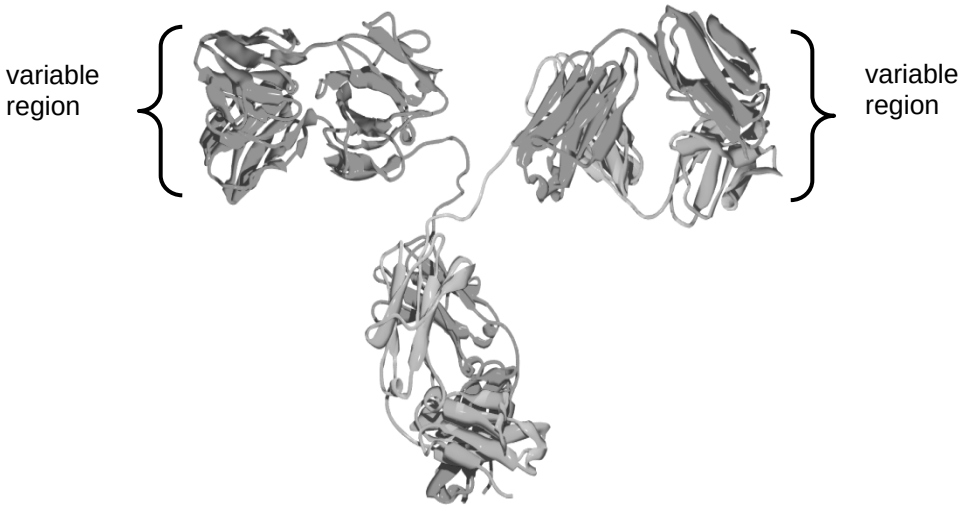


Fig. 9.2

(b) Describe how the antibody is folded from linear polypeptide chains. [4]

A study investigated active and passive immunity to tetanus toxin. One person, **G**, was injected with antibodies to the tetanus toxin. Another person, **H**, was injected with the vaccine for tetanus and produced antibodies as a result. Blood samples were taken from **G** and **H** at regular intervals over the following weeks and analysed for antibodies against tetanus.

The results of the study are shown in Fig. 9.3.

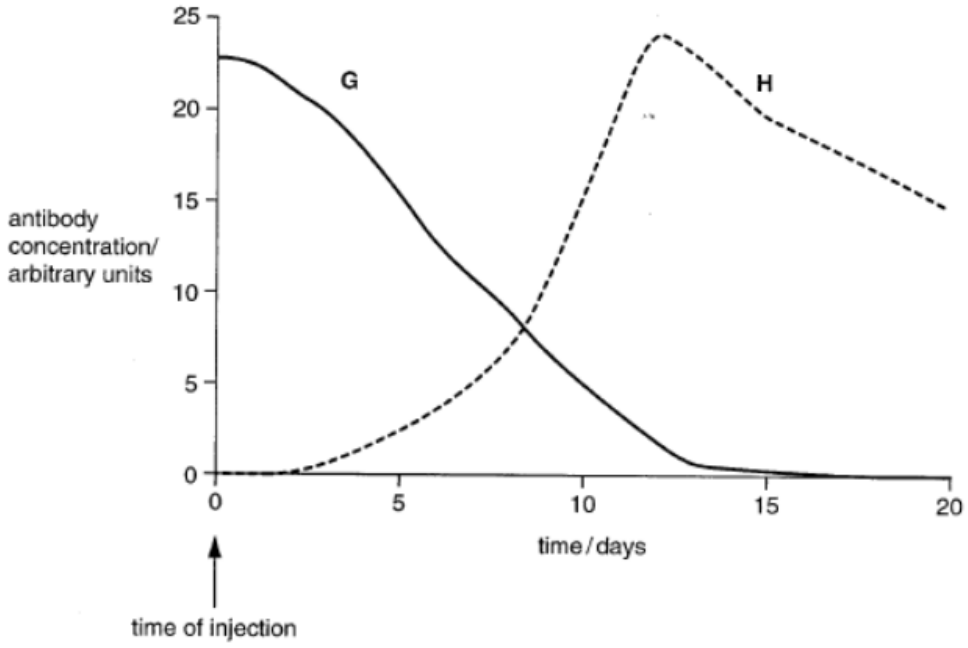


Fig. 9.3

(c) Explain why the type of immunity gained by **G** is described as passive immunity. [2]

(d) With reference to Fig. 9.1 and Fig. 9.3, explain why there is a slow increase in antibody concentration in the curve for **H**. [2]

(e) Explain why person **H** is considered to be better protected against future exposure to the tetanus toxin, compared to person **G**. [2]

[Total: 12]