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.

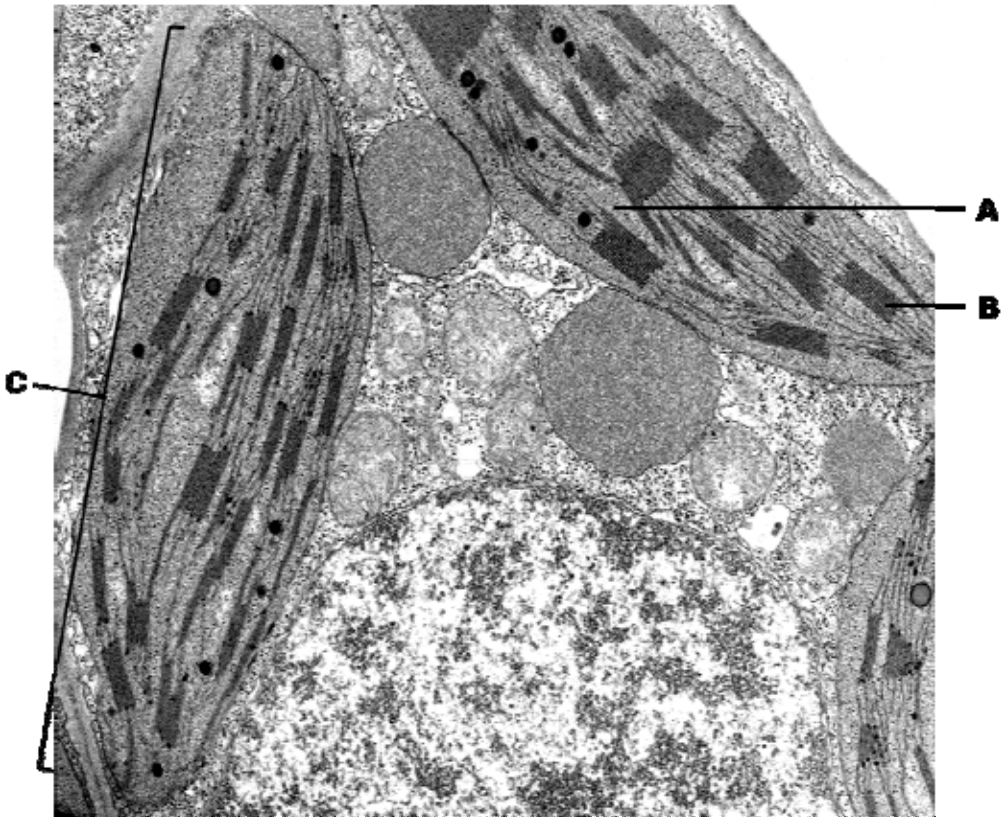


Fig. 1.1

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

1. **Stroma (of chloroplast);;**
2. **Site of light independent reactions / Calvin cycle;;**

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

1. **(S) Large surface area of thylakoid membrane;**
2. **(F) Holds photosystems for light absorption / ETC for transfer of electrons;**
3. **(S) Thylakoid membrane impermeable to H^+;**
4. **(F) Allows proton gradient to be generated (for chemiosmosis);**
5. **(S) Contains ATP synthase;**
6. **(F) For phosphorylation of ADP to ATP / ATP synthesis.**

Cyanobacteria are prokaryotic cells that are capable of carrying out photosynthesis. The

structure of a cyanobacteria is shown in Fig. 1.2.

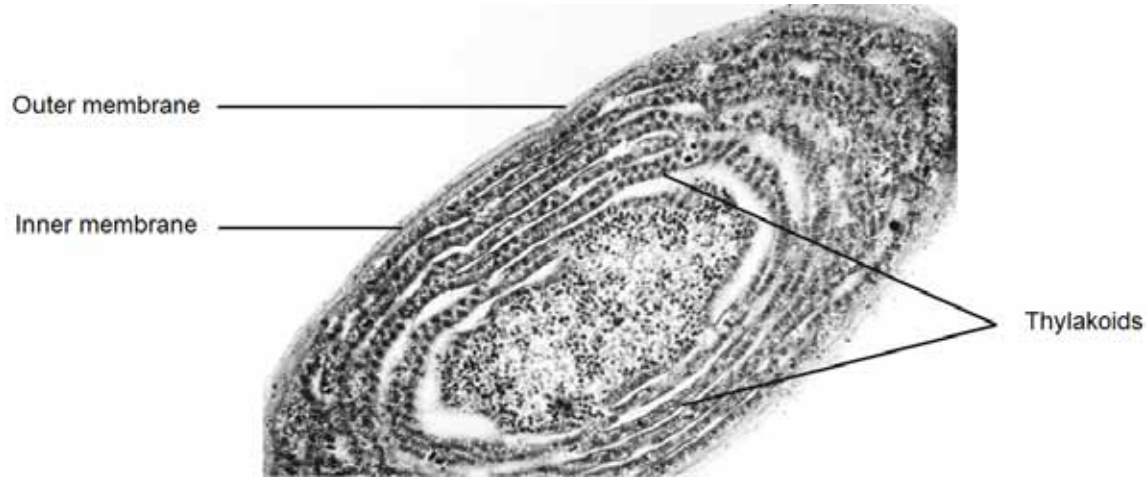


Fig. 1.2

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

Similarities (1 max)

1. **Both are bound by two membranes;;**
2. **Both have thylakoids;;**

Differences (1 max)

Feature	**Chloroplast**	**Cyanobacteria**
Grana	**Present**	**Absent;;**
Intergranal lamellae	**Present**	**Absent;;**

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]

1. **contains circular DNA;;**
2. **has 70S ribosomes;;**
3. **divides via binary fission;;**

[Total: 9]

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

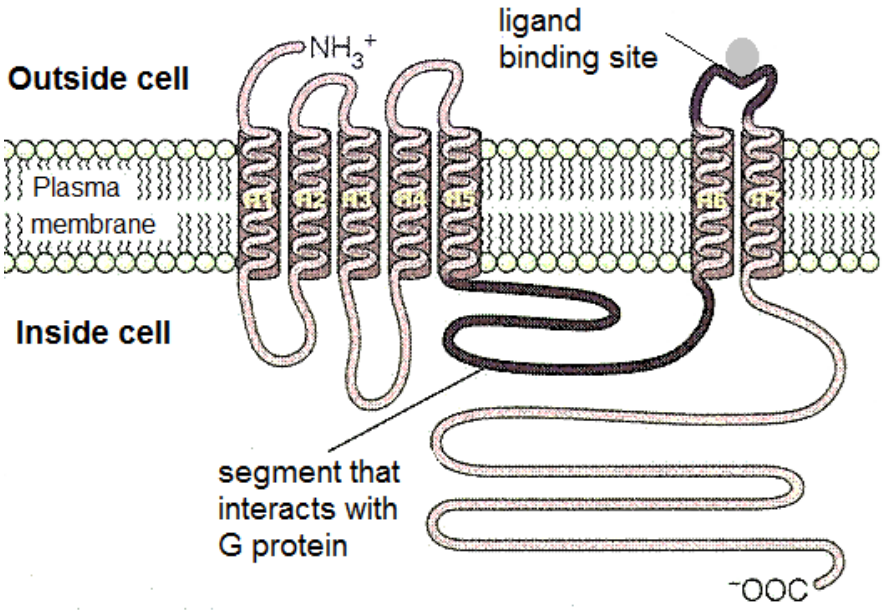


Fig. 2.1

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

1. **(S) The seven α-helices are mainly hydrophobic;**
2. **(S) can interact with the hydrocarbon tails of phospholipids via hydrophobic interaction;**
3. **(F) embeds GPCR in the cell surface membrane;**
4. **(S) has an extracellular binding site;**
5. **(F) for binding to complementary shape ligand;**
6. **(S) has an intracellular binding site / segment;**
7. **(F) (for binding to G protein) for activation of G protein;**
8. **(F) to transduce extracellular signals for activation of intracellular proteins / signal transduction;**

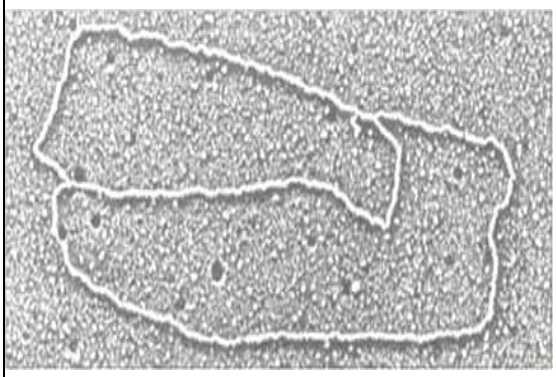
One of the cellular events resulting from glucagon binding to GPCR, shown in Fig. 2.1, is the activation of glycogen phosphorylase which breaks down glycogen to glucose.

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

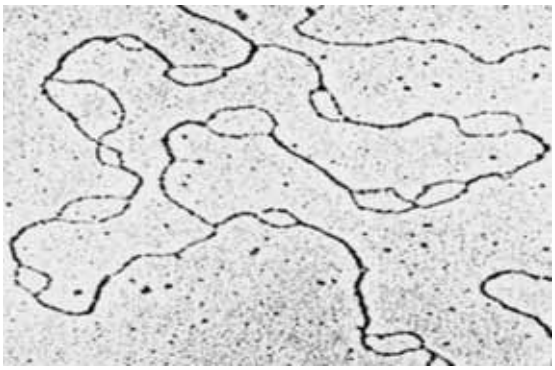
1. **(binding of glucagon to ligand binding site on GPCR) causes conformation change of receptor;**
2. **GTP displace GDP in G protein;**
3. **activating it;**

			4. and move along membrane to activate adenylyl cyclase; **5. Catalyse conversion of ATP to cAMP;** **6. Activation of protein kinase A;** **7. Activate phosphorylation cascade;**	
		(ii)	Explain why liver cells store glucose in the form of glycogen.	[3]
			1. (large molecule) insoluble in water and will not exert osmotic or chemical influence on the cell;; **2. anomeric carbon is involved in glycosidic bond making glycogen stable and unreactive;;** **3. extensively branched, hence is compact in shape;;**	
	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]
			1. Net movement of glucose; **2. down concentration gradient;** **3. via channel /carrier protein;** **4. without additional investment of energy;**	
		(ii)	Explain why glucose transporters are necessary to facilitate this process.	[2]
			1. glucose is polar;; **2. cannot diffuse across <u>hydrophobic</u> core;** **3. of membrane/phospholipid layer;**	
				[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]

1. **Single origin of replication in bacterium but multiple origin of replication in mammalian cell;; Accept: multiple replication sites / bubbles**
2. **Larger / longer DNA, speed up replication;;**

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

1. **Parental DNA strands separate / ref. to H bonds break;**
2. **Both strands acts as a <u>template</u> for synthesis of daughter strand;**
3. **Each daughter DNA molecule consists of one parental strand and one newly synthesised strand of DNA;;**

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.

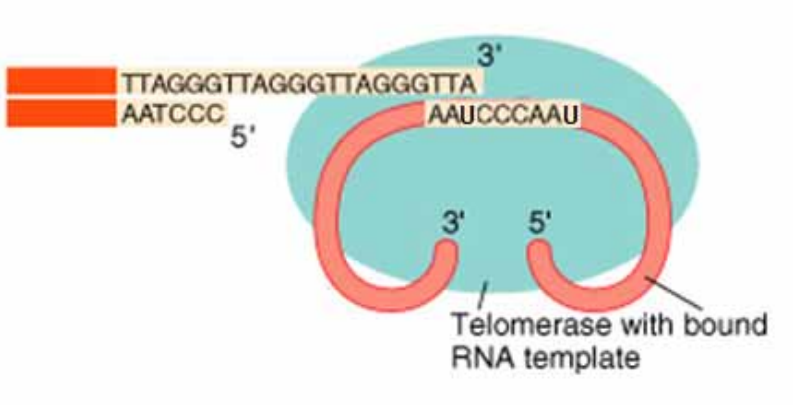


Fig. 3.2

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

1. **due to the specificity of DNA polymerase;**
2. **the RNA primers complementary to the 3' ends (of the template DNA);**
3. **cannot be replaced after their removal;**
4. **without a 3' hydroxyl group on the DNA strand for DNA polymerase to add nucleotides to;**

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

Feature	Lengthening of DNA	Transcription
Template	RNA	DNA
Monomers	DNA nucleotides	RNA nucleotides
Enzyme involved	Telomerase	RNA polymerase
Product synthesised	DNA	RNA

1 mark for each comparative statement

Any two

[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.

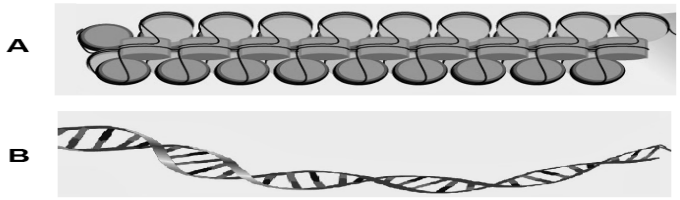


Fig. 4.1

(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]

1. **DNA coils around histone octamer;**
2. **Forming 'beads on a string' structure' / 10 nm chromatin fibre / nucleosome fibre;**
3. **Histone H1 further interacts;**
4. **with linker DNA; giving rise to the structure**

(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]

1. **Downregulation of transcription;;**
2. **DNA is condensed / highly folded;**
3. **Promoter not accessible;**
4. **for binding of RNA polymerase / transcription factors;**
5. **to <u>initiate</u> transcription;**

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-	De-	Ubiquitin not	Ubiquitin not	Ubiquitin not	Remains

	phosphorylated	phosphorylated	attached	attached	attached	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]

Post-translation;;

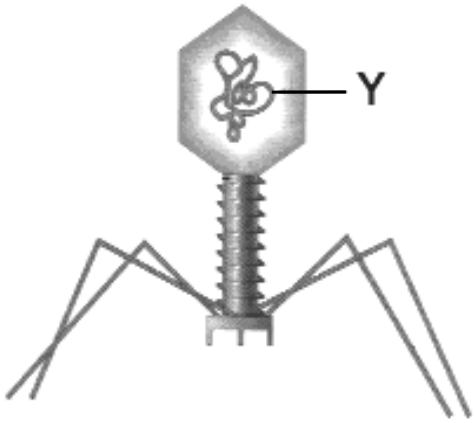
(ii) describe the events at the selected amino acids that triggers the degradation of Huntingtin proteins. [2]

1. **Phosphorylation of serine residues;**
2. **at position 13 and 16;**
3. **ubiquitination of lysine residues;**
4. **at positions 6, 9, and 15;**

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

1. **Ubiquitin marks the mutant Huntingtin protein for degradation;;**
2. **Proteasomes recognises ubiquitin (tag) on protein;**
3. **and hydrolyses / breaks down protein into peptide / amino acids;**

[Total: 10]

5	Fig. 5.1 shows the structure of a T4 virus.  **Fig. 5.1**		
	(a)	Identify structure **Y**.	[1]
		Double-stranded deoxyribonucleic acid;;	
	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]
		1. lacks a named enzyme (e.g. RNA polymerase / DNA polymerase);; **2. lacks a named organelle (e.g. golgi apparatus for protein modification / RER for protein synthesis);;** **3. lacks a named molecule for protein synthesis / DNA replication;;** **4. lacks a named energy resources, e.g. ATP;;**	
	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.		

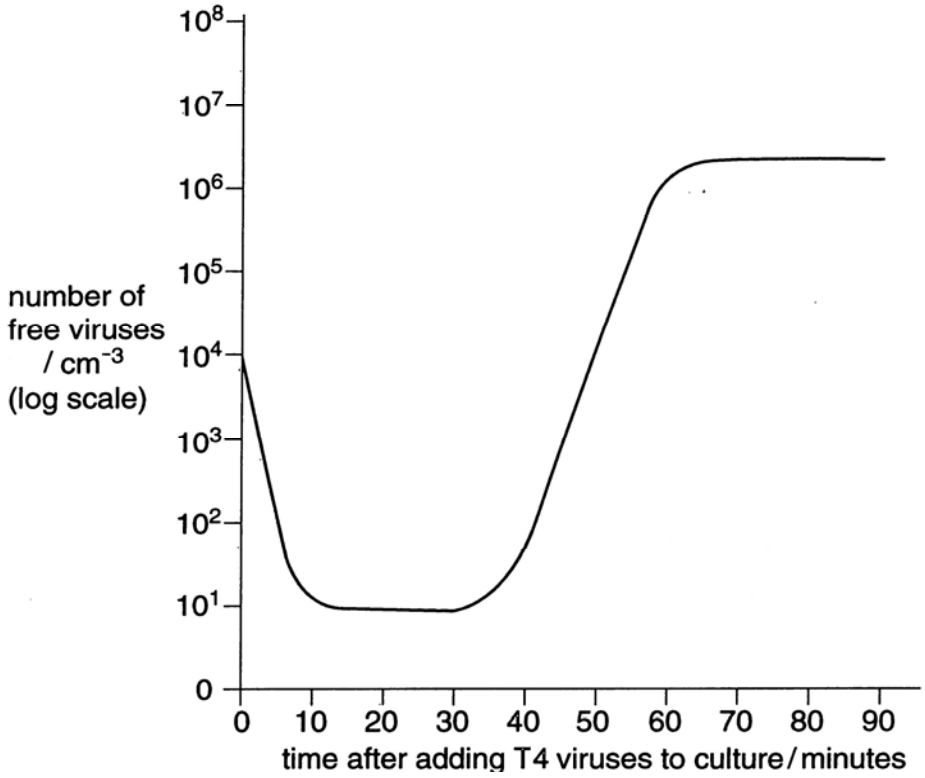


Fig. 5.2

	(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]
			1. number of free viruses decreases from 10^4 to 10^1 cm^{-3};; **2. Due to attachment of viruses on the *E. coli*;;** **Reject: viruses enter host cell**	
		(ii)	between 30 and 60 minutes.	[3]
			1. number of free viruses increases from 10^1 to 10^6 cm^{-3};; **2. due to lysis of host cell to release viruses;;** **3. (increase in number of viruses from 10^4 to 10^6) due to multiplication of viruses;;**	

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

T4 are mixed with B^+ cells

↓

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

↓

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

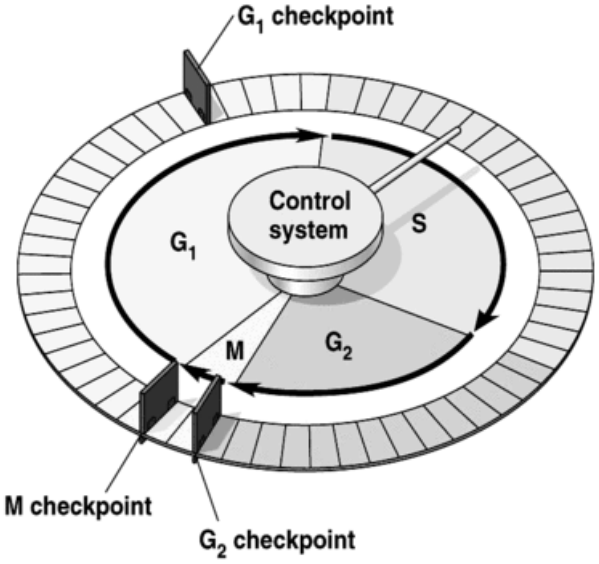
↓

Growth observed on medium

Fig. 5.3

	(d)	**(i)**	Explain the observations made by the scientist.	[3]
			1. (Generalised) transduction; **2. T4 infects B^+ cell;** **3. Fragment of B^+ DNA confers ability to produce lysine;** **4. are accidentally packaged into phage capsid;** **5. Upon release from B^+ cell, transducing phage <u>infects</u> new B^- cell;** **6. B^+ DNA incorporated into B^- DNA (via homologous exchange);** **Penalise 1 mark for lack of contextualisation**	
		(ii)	Suggest **one** other potential benefit of the process mentioned in **(d)(i)** for the recipient bacteria.	[1]
			1. Develop antibiotic resistance/ xenobiotic (chemical) resistance;; **2. Ability to utilise a new metabolite;;**	
				[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]

1. **Interphase;;**
2. **Accumulation of energy stores / ATP;**
3. **Synthesis of proteins;**
4. **Synthesis of (cytoplasmic) organelles;**
5. **Replication of centrioles;**
6. **DNA replication;**

   R: synthesis of nucleic acids

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

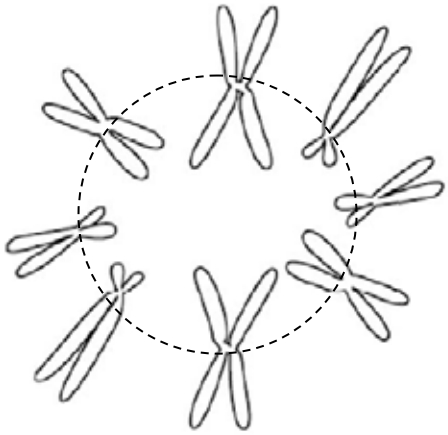


Fig. 6.2

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

Type: mitosis;

Stage: metaphase;

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

1. **Chromosomes lined up at metaphase plate singly (∴not meiosis I);;**
2. **(4) pairs of homologous chromosome present (∴ not meiosis II);;**

(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	**8;**	170
Metaphase of mitosis	**8;**	**170;**
Telophase of mitosis	**8;**	**85;**

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]
		1. Normal genes; **2. which codes for a protein;** **3. that promotes <u>normal</u> cell division;**	
	(e)	Explain how a mutant Ras protein may lead to cancer.	[3]
		1. Hyperactive / degradation-resistant Ras protein;; **2. relays <u>signal</u> from growth factor / triggers <u>kinase cascade</u>;** **3. in the absence of growth factor;** **4. Resulting in proteins that stimulate cell cycle;** **5. thus uncontrolled cell division;**	
	(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]
		1. unspecialised cell / pluripotent; **2. can divide and grow <u>indefinitely</u>;** **3. differentiate into any cell type except those that form the placenta and the umbilical cord under appropriate conditions;** **4. found l inner cell mass of blastocyst;**	
			[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]

BbEe / BBEe;;

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

F_1 phenotypes Black x Golden
F_1 cross BbEe x bbee ;

F_1 gametes (BE) (Be) (bE) (be) ; (be) ;

Random Fertilization (as shown in the Punnett Square)

	(BE)	(Be)	(bE)	(be)
(be)	BbEe Black	Bbee Golden	bbEe Brown	bbee; Golden;

Offspring phenotypic ratio 1 Black : 2 Golden : 1 Brown ;

OR

F_1 phenotypes Black x Golden
F_1 cross BBEe x bbee ;

F_1 gametes (BE) (Be) ; (be) ;

Random Fertilization (as shown in the Punnett Square)

	(BE)	(Be)
(be)	BbEe Black	Bbee Golden

Offspring phenotypic ratio 1 Black : 1 Golden;

	(c)		Name and describe the type of interaction between the gene loci.	[3]
			1. (recessive) Epistasis;; **2. ee is epistastic over the B/b locus;** **3. gene E encodes for enzyme E;** **4. converts golden precursor;** **5. to brown pigment;** **6. gene B encodes enzyme B;** **7. converts brown pigment to black pigment;**	

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

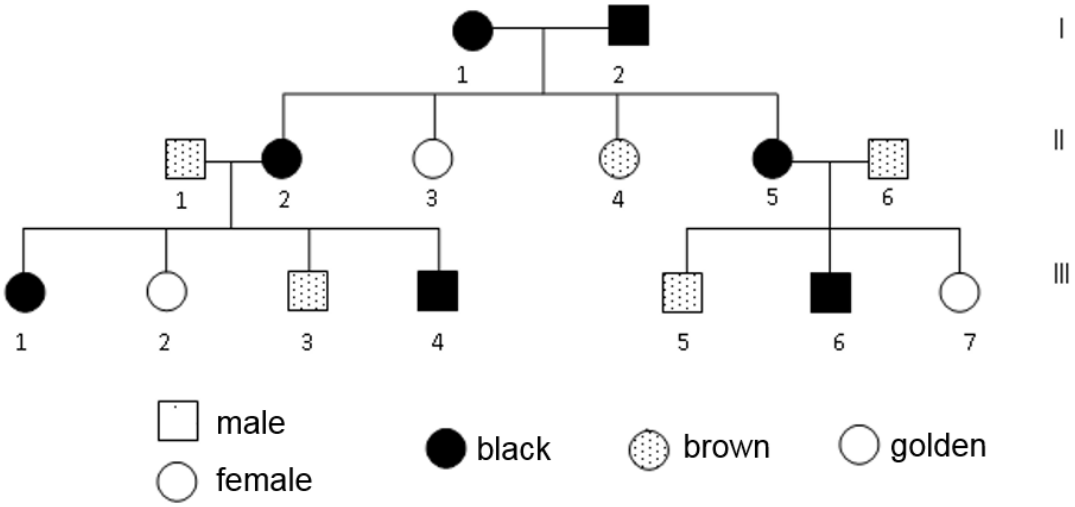
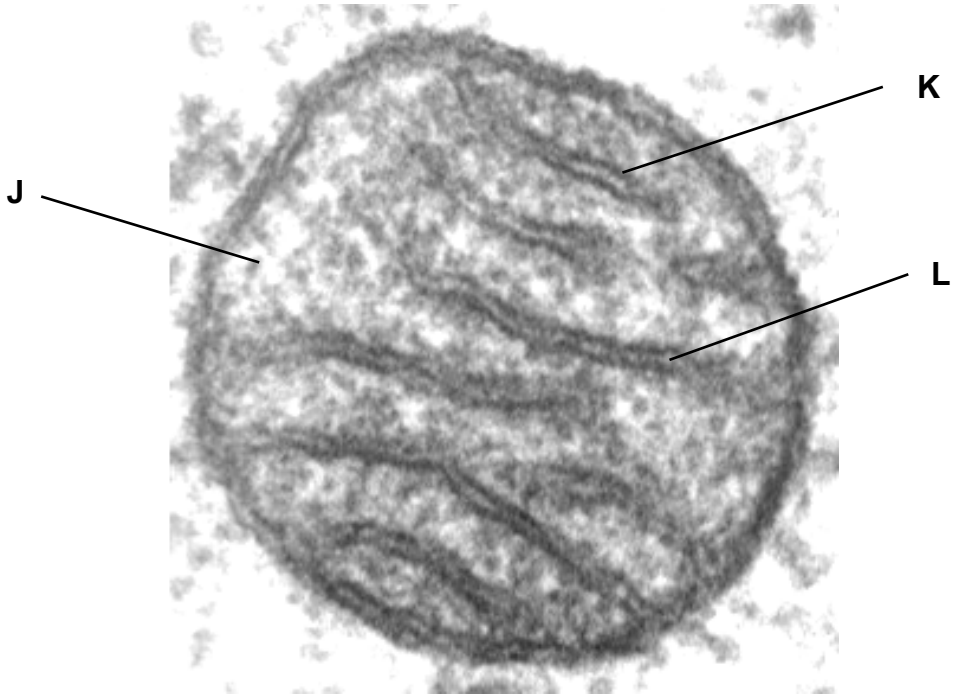


Fig. 7.1

	(d)	**(i)**	State the genotype of individual II-1.	[1]
			bbEe;;	
		(ii)	Explain your answer in **d(i)**.	[2]
			1. Individual II-1 is brown, genotype must be bbE_;; **2. Individual II-1 has golden offspring, must be heterozygous at E/e locus;;**	
				[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	**(mitochondrial) Matrix;**	
			K	**inner (mitochondrial) membrane / crista;**	
		(ii)	Describe how structure **J** is adapted to its function.		[1]
			Matrix contains enzymes for Krebs cycle / link reaction;;		
	(b)	**(i)**	State the role of high concentration of protons at **L**.		[1]
			It acts as a source of potential energy for the synthesis of ATP by ATP synthase;;		
		(ii)	Explain how the high concentration of protons is generated at **L**.		[3]
			1. electrons from NADH / $FADH_2$; **2. passes along a chain of electron carriers (releasing energy in a series of small steps);** **3. <u>free</u> energy released;** **4. is used to pump protons;** **5. from matrix into intermembrane space;**		

6. inner mitochondrial membrane is impermeable to ions;

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]

1. no glycolytic enzymes in mitochondria;
2. glycolysis does not occur in the mitochondria / glycolysis can only occur in the cytosol;
3. glucose cannot be oxidised to form pyruvate;
4. pyruvate can enter mitochondria but glucose cannot;
5. CO_2 produced by decarboxylation in link reaction;
6. and Krebs cycle;

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

1. Chemical M only block ATP synthase so no phosphorylation of ADP/no flow of H^+ down concentration gradient (through ATP synthase);;
2. Chemical M does not affect ETC to transfer electrons to oxygen;;

[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.

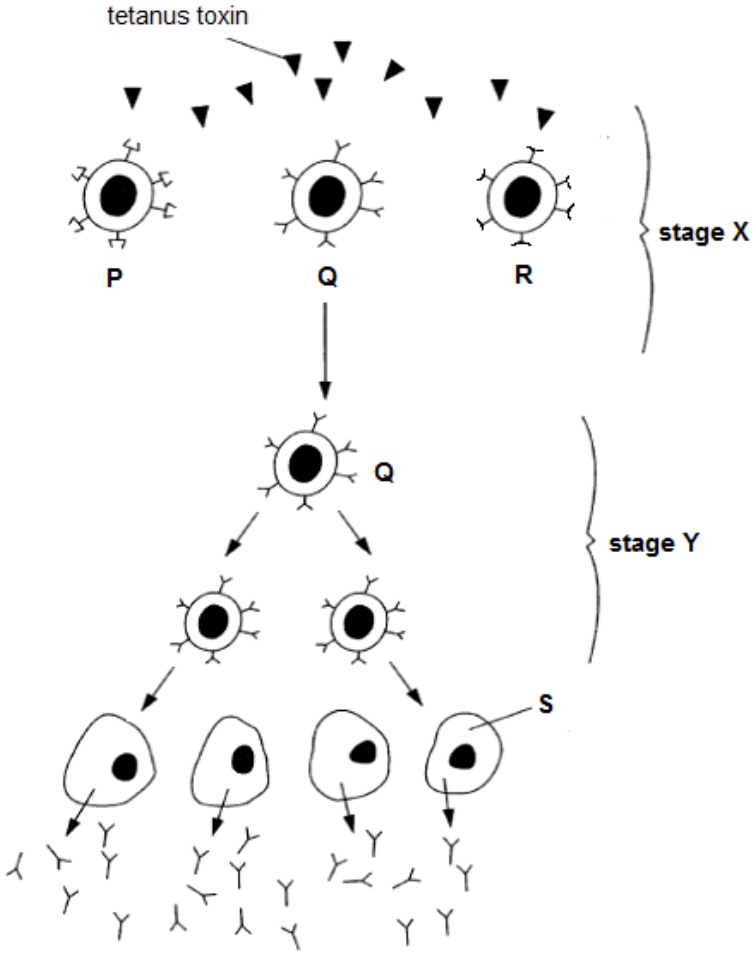


Fig. 9.1

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

X:

1. **Only Q has receptor / clonal selection;**
2. **Receptor and toxin have complementary shape;;**

Y:

1. **Q undergoes clonal expansion / form clone / divide / increase number / mitosis;**

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

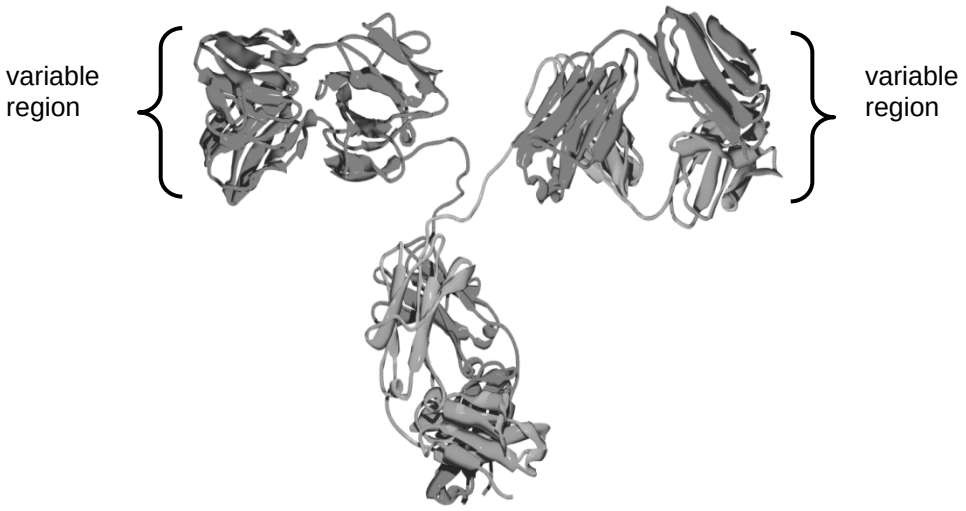


Fig. 9.2

	(b)	Describe how the antibody is folded from linear polypeptide chains.	[4]
		1. Localised folds; **A: (H) bonds between CO and NH groups** **2. along the polypeptide backbone;** **3. give rise to α-helix;** **4. and β-pleated sheets;** **5. Interactions between R groups of amino acid residues;** **6. bends the (secondary) structure into tertiary / precise / compact / globular shape;** **7. (quaternary structure) consist of 4 polypeptide chains;** **8. 2 heavy chains and 2 light chains;**	

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.

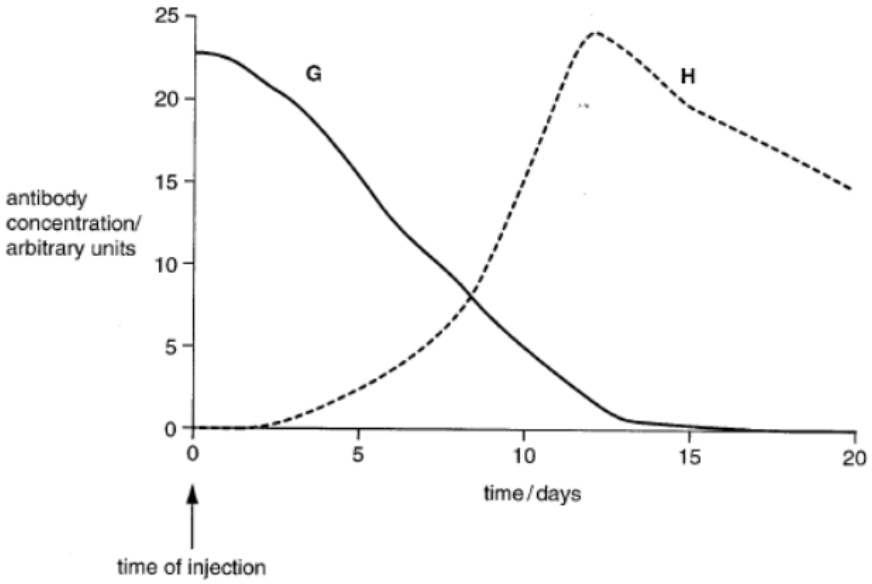


Fig. 9.3

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

1. **No immune response elicited;**
2. **Antibodies, not made / come from other source;**
3. **High concentration / figure from graph, immediately / after injection / on Day 0-1;**
4. **Antibody concentration fall / AW;**
5. **Does not last long / only approximately 2 weeks / temporary;**

(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]

Time needed for

1. **Antigen presentation;;**
2. **Activation of T helper cells;;**
3. **Clonal expansion / mitosis / AW;;**
4. **Differentiation of B cells into plasma cells;;**
5. **Antibody synthesis / release from plasma cell;;**

Any two

	(e)	Explain why person **H** is considered to be better protected against future exposure to the tetanus toxin, compared to person **G**.		[2]
		1. Person H has immunological memory / memory cells; **2. able to elicit a secondary response;** **3. which is rapid;** **4. and allows a larger production of antibody;**		
				[Total: 12]