

Cambridge International AS & A Level

BIOLOGY (9700) PAPER 2

Past Paper Questions By Topic
+ Answer Scheme

2015 - 2020

Complete Syllabus



Chapter 11

Immunity



11.1 The immune system

200. 9700_s19_qp_22 Q: 3

Fig. 3.1 is a photomicrograph of human blood cells from a healthy individual who lives at sea level. The cells labelled **C**, **D** and **E** are white blood cells.

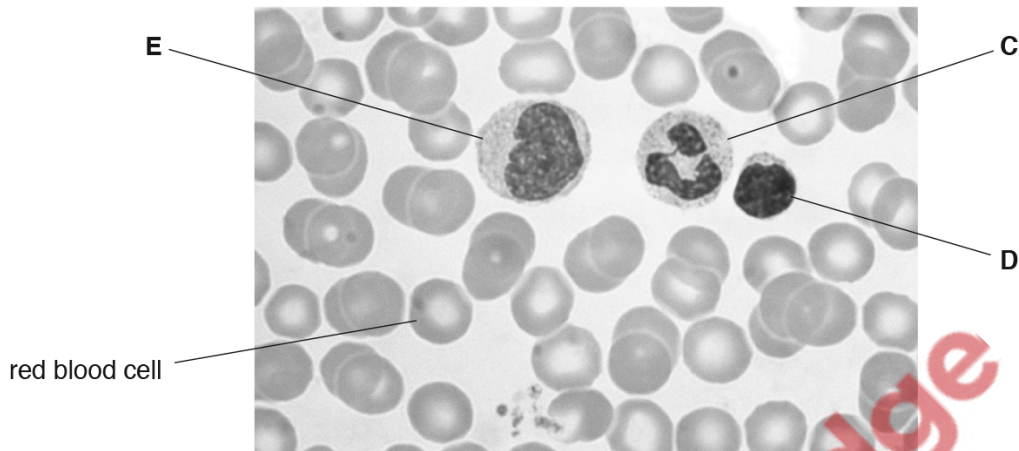


Fig. 3.1

(a) Name cells **C**, **D** and **E**.

C

D

E [3]

(b) In humans, an increase in the white blood cell count can be associated with leukaemias and with infectious diseases, such as measles.

Chronic lymphocytic leukaemia (CLL) is a type of cancer that starts in the bone marrow. In the early stages, many people with CLL feel well. The disease is sometimes diagnosed by chance during a routine blood analysis, when a high white blood cell count is noticed. Many of these white blood cells are only partially mature.

(i) Suggest why **CLL** starts in the bone marrow and **not** in any other location in the body.

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..... [2]

- (ii) Explain why a high white blood cell count is a feature of measles and of CLL.

measles

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CLL

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- (c) Most of the oxygen that enters the mammalian circulatory system is transported by red blood cells.

- (i) Describe **and** explain the passage of oxygen across the cell surface membrane of the red blood cell.

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..... [2]

- (ii) At a high altitude, the partial pressure of oxygen in the atmosphere is lower than at sea level. If a person travels from low altitude to high altitude and remains there for a few weeks, the red blood cell count increases.

Explain why the body needs to respond to high altitude by increasing the number of red blood cells.

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(d) Polypeptide synthesis occurs before a red blood cell is released into the circulation.

The *HBB* gene codes for the β -globin polypeptide of haemoglobin.

There are two alleles of *HBB*, known as Hb^A and Hb^S .

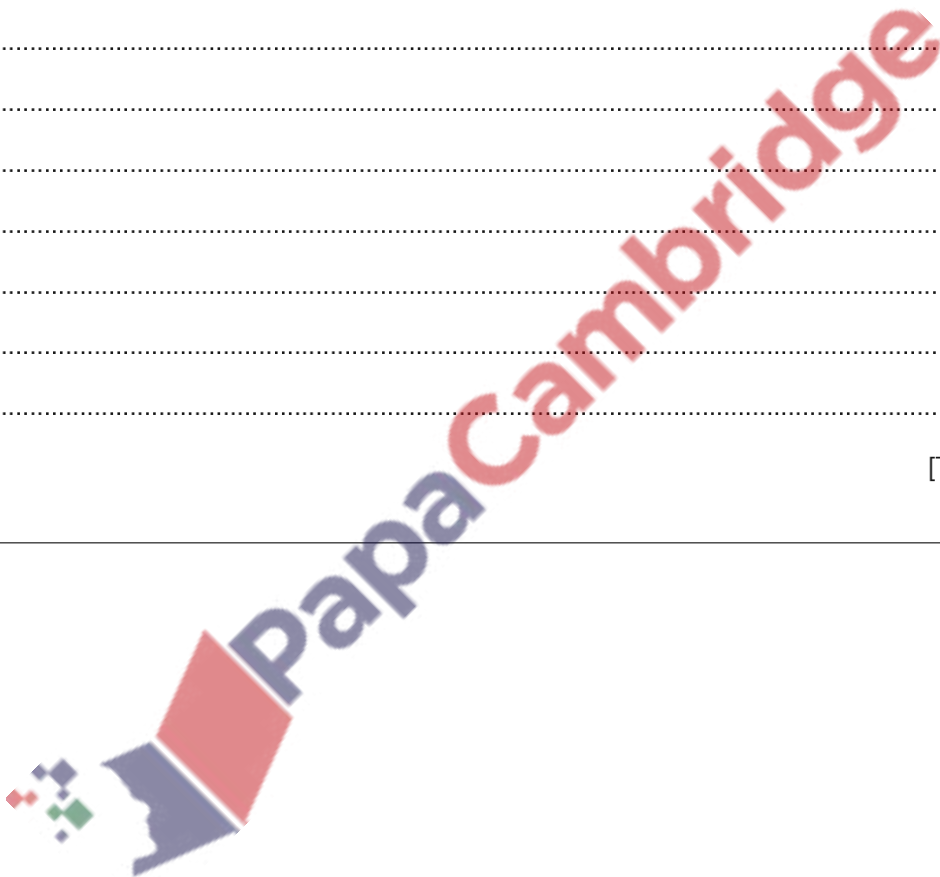
Describe the difference between the Hb^A allele and the Hb^S allele **and** state how this difference affects:

- the β -globin polypeptide
- the haemoglobin molecule.

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[Total: 17]



- (c) The World Health Organization has suggested that people with HIV/AIDS take a longer time to recover from cholera and are at an increased risk of death from cholera.

The human immunodeficiency virus (HIV) only infects certain types of cell. These cells have CD4 receptor proteins in their cell surface membrane. Helper T-lymphocytes have CD4 receptor proteins.

- (i) Fig. 3.1 is a diagram of HIV showing the glycoprotein gp120.

This glycoprotein is embedded in a membrane envelope which surrounds the viral protein coat.

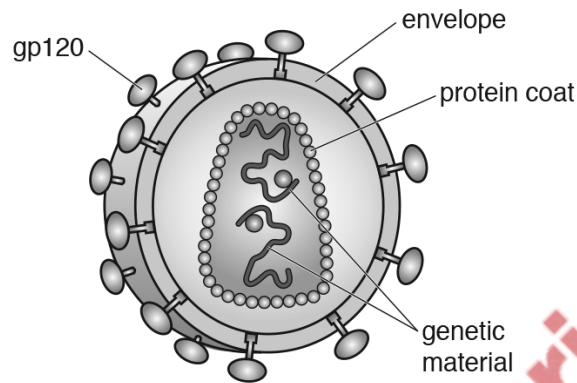


Fig. 3.1

The glycoprotein gp120 is important in allowing HIV to only infect certain types of cell.

Suggest the role of gp120.

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[2]



- (ii) People with HIV/AIDS have a low helper T-lymphocyte count.

Explain how a low helper T-lymphocyte count could reduce the body's ability to produce antibodies against *V. cholerae*.

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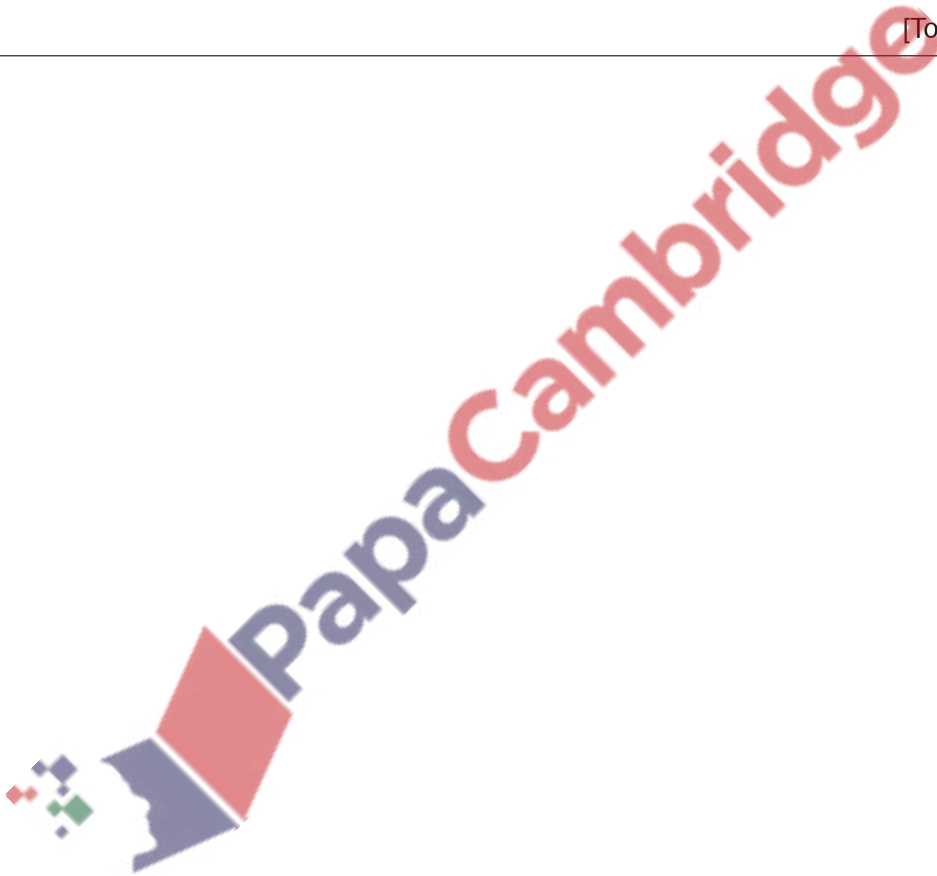
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[2]

[Total: 13]



202. 9700_m18_qp_22 Q: 2

The main cause of tuberculosis (TB) in humans is the bacterium *Mycobacterium tuberculosis*. Most cases of the disease involve the lungs. The bacterium can enter cells and remain inactive in a latent (dormant) state. However, the bacterium can become active to produce symptoms of the disease.

In a person with active TB, the pathogen can be present in airborne droplets that are exhaled. Generally, a healthy person who inhales these droplets has effective defence mechanisms in the gas exchange system to prevent infection.

(a) One example of a defence mechanism against pathogens in the gas exchange system involves the action of macrophages.

(i) State the location in the body where macrophages have their origin.

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(ii) Describe the mode of action of a macrophage.

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(iii) It is sometimes possible for *M. tuberculosis* to survive within macrophages. Suggest **one** way in which *M. tuberculosis* may survive within a macrophage.

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- (b) A healthy person has other defence mechanisms in the gas exchange system to prevent bacteria entering cells.

Describe these defence mechanisms **and** explain how bacteria in inhaled air are prevented from entering cells of the gas exchange system.

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- (c) In people with a weakened immune system, *M. tuberculosis* can infect other organs and tissues, such as the kidneys and joints.

Suggest how the bacteria may spread from the lungs to other organs.

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- (d) TB in humans can be caused by another species of bacterium, *M. bovis*.

State the mode of transmission of this pathogen to humans.

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- (e) The standard treatment for TB continues for six months and initially involves the use of four different antibiotics.

If no antibiotic resistance is detected, the treatment is reduced to two of the four antibiotics. The two antibiotics used are rifampicin and isoniazid.

Suggest the benefits of beginning the treatment with four different antibiotics.

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Multidrug-resistant TB (MDR-TB) occurs if resistance develops to rifampicin and isoniazid.

The treatment for MDR-TB can last up to 30 months and involves different antibiotics to the standard treatment.

Table 2.1 shows the number of reported cases of TB and MDR-TB in the South-East Asia region between 2005 and 2014, as published by the World Health Organization (WHO).

Table 2.1

year	total number of reported cases of TB	total number of reported cases of MDR-TB
2005	1 947 603	68
2006	2 104 673	779
2007	2 202 149	918
2008	2 287 803	1 717
2009	2 328 230	2 560
2010	2 332 779	4 263
2011	2 358 127	6 615
2012	2 331 455	14 957
2013	2 297 033	18 384
2014	2 580 605	17 386

(f) State the trends shown in Table 2.1.

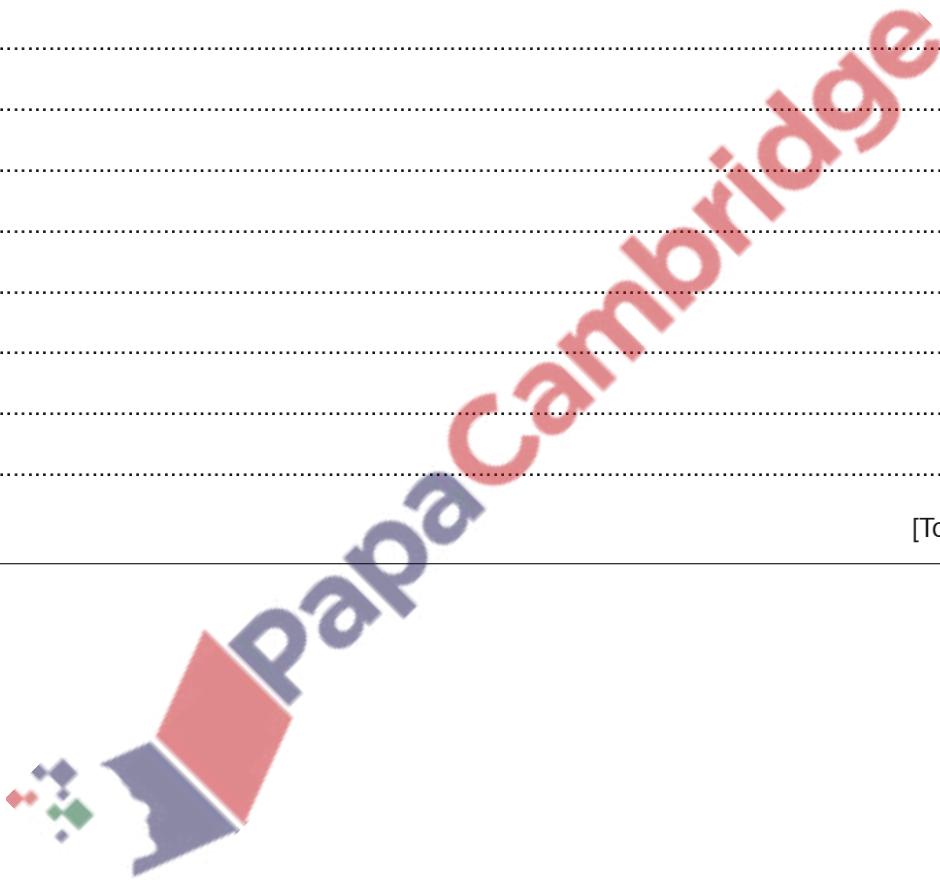
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(g) TB is a disease of global importance.

Discuss the factors influencing the trends shown in Table 2.1.

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[Total: 17]



203. 9700_s18_qp_21 Q: 5

- (a) The toxins released by some pathogenic bacteria can be altered chemically so that they are harmless. These harmless toxins are called toxoids.

Toxoids are used in vaccines to provide protection against some infectious diseases.

Describe the response of the immune system to the injection of a toxoid.

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- (b) Myasthenia gravis (MG) is described as an autoimmune disease. It is a long-term condition that results from a failure of the immune system.

(i) Explain why MG is known as an autoimmune disease.

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(ii) Suggest why MG is a long-term condition.

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[Total: 10]

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204. 9700_s18_qp_22 Q: 3

Bacteria may be classified according to differences in cell wall structure. The differences are shown by using the Gram stain.

- A Gram-positive bacterium has a cell wall mainly composed of a thick layer of peptidoglycan (murein).
- A Gram-negative bacterium has a more complex cell wall. This wall is composed of a much thinner layer of peptidoglycan and an outer layer known as the outer membrane.

Escherichia coli is a Gram-negative bacterium.

Fig. 3.1 is a diagram through the cell surface membrane **and** the cell wall of *E. coli*.

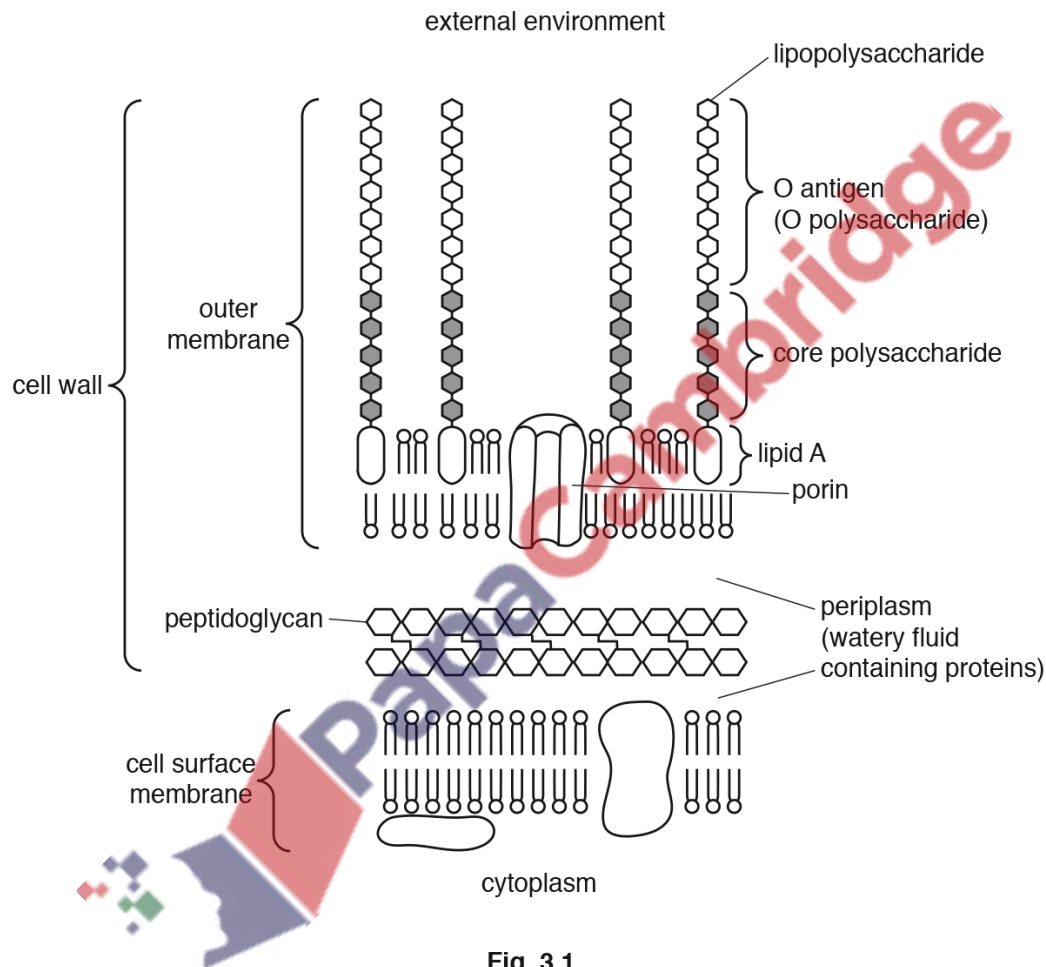


Fig. 3.1

- (a) The antibiotic penicillin kills bacteria by causing them to lyse (burst). It is more effective in treating diseases caused by Gram-positive bacteria than diseases caused by Gram-negative bacteria.

Outline how penicillin acts on bacteria **and** use Fig. 3.1 to suggest why penicillin has little or no effect at treating diseases caused by Gram-negative bacteria, such as some strains of *E. coli*.

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- (b) The outer membrane contains transport proteins called OmpF porins. These porins allow the passive movement of water, ions and small, polar molecules across the outer membrane. Each OmpF porin is formed from three identical polypeptides.

(i) Explain what is meant by the term *passive*.

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(ii) Suggest **and** explain the features of an OmpF porin as a membrane transport protein.

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- (iii) *E. coli* can regulate the number of OmpF porins in the outer membrane to adapt to changing conditions. One control mechanism used by *E. coli* involves the production of a small mRNA molecule known as micF.

MicF binds to the part of the mRNA molecule containing the START codon for the OmpF polypeptide.

Suggest **and** explain how the presence of micF prevents production of OmpF porins.

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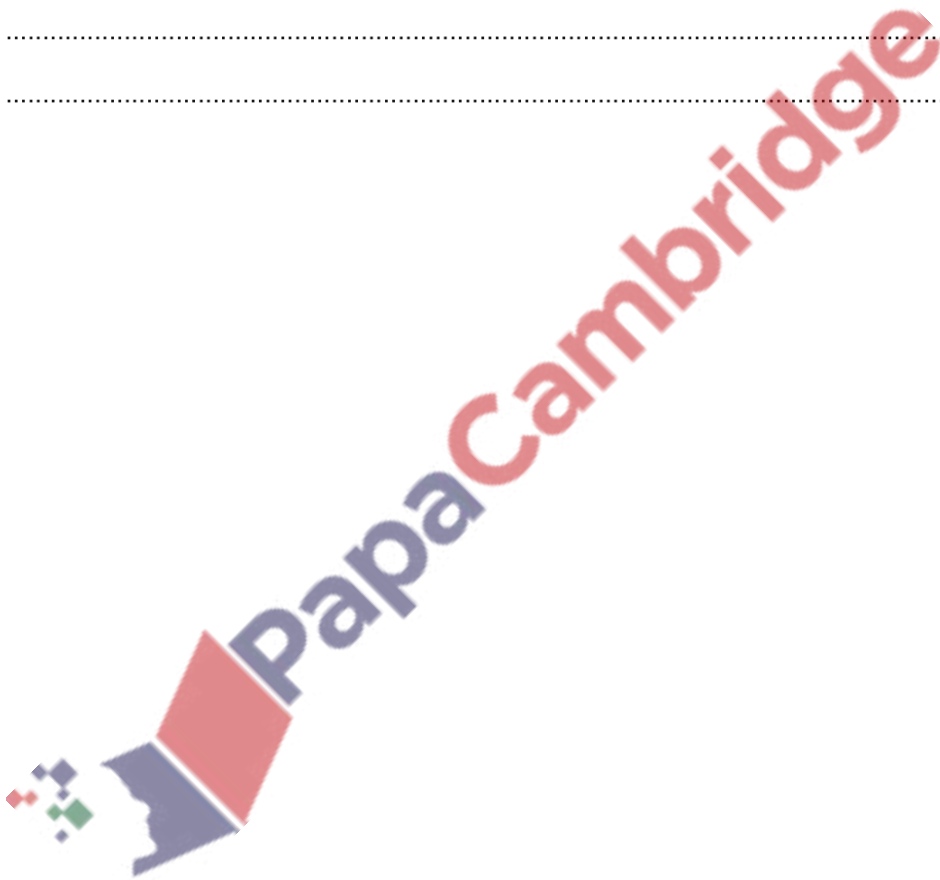
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- (c) Fig. 3.1 shows that the outer membrane of the cell wall of *E. coli* contains lipopolysaccharides. These are not present in the cell surface membrane. Each lipopolysaccharide (LPS) consists of a lipid and a polysaccharide portion.

The O antigen is the outer part of the polysaccharide portion of the LPS. It faces the aqueous external environment.

- (i) Define the term *polysaccharide*.

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- (ii) Some strains of *E. coli* are pathogenic. Different pathogenic strains have different O antigens.

Suggest **and** explain why infection with one pathogenic strain of *E. coli* does not provide immunity to a different pathogenic strain.

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[Total: 15]



205. 9700_w17_qp_22 Q: 4

Fig. 4.1 is a transmission electron micrograph of the bacterium that causes cholera, *Vibrio cholerae*.

The flagellum shown in Fig. 4.1 allows movement of the bacterium within the gut and may also function to help it to bind to an intestinal epithelial cell. The organism does not enter the cell but the toxin it releases can enter and cause damage. Large quantities of water, chloride ions and sodium ions are lost from the cell.



Fig. 4.1

People with symptoms of cholera have severe watery diarrhoea and as a result can become very dehydrated.

(a) Explain how a loss of chloride ions and sodium ions from the intestinal epithelial cell will cause a loss of water from the cell.

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- (b) The main treatment for cholera is oral rehydration therapy (ORT) using oral rehydration salts (ORS). This involves drinking a solution of electrolytes (mineral ions) and glucose.

Fig. 4.2 summarises the movement of glucose and sodium ions across an intestinal epithelial cell.

Fig. 4.2 includes three different types of cell surface membrane proteins:

- SGLT1 is a cotransporter protein
- GLUT2 and Na^+/K^+ pump are two types of carrier protein.

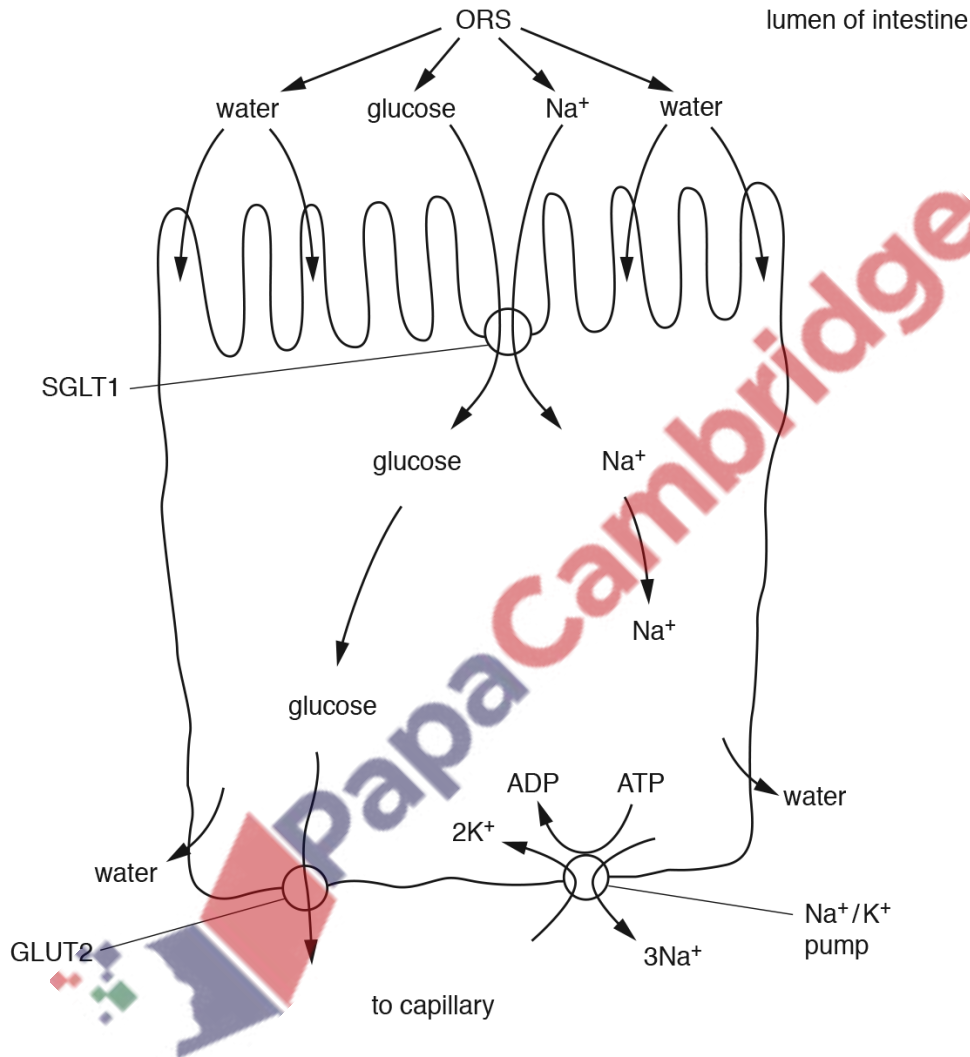


Fig. 4.2

(e) A study was carried out to compare the effectiveness of the antibiotic tetracycline in the treatment of 118 patients with cholera. The patients were divided into four different treatment groups:

- Group **A**, given one dose of 1 g tetracycline
- Group **B**, given one dose of 2 g tetracycline
- Group **C**, given a multiple dose (one dose of 500 mg tetracycline every 6 hours for 24 hours)
- Group **D**, no antibiotic given.

Following treatment, the volume of diarrhoea collected from each patient was measured every 16 hours for 128 hours. Fig. 4.3 shows the mean volume collected for each group.

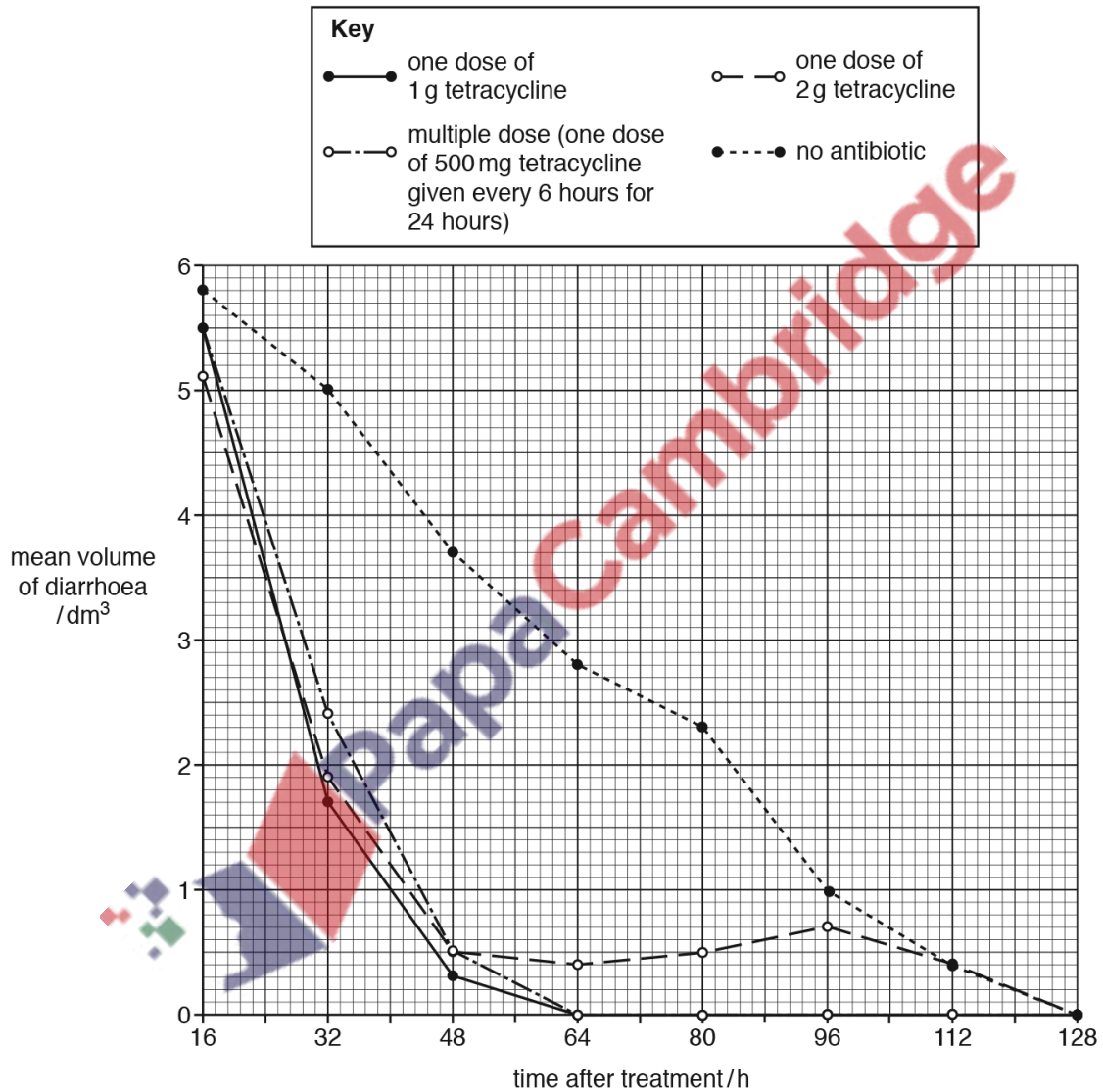


Fig. 4.3

- (g) Most people who have recovered from cholera rarely become ill again from the disease. In these people, antibodies have been identified that will bind either to the cholera toxin, or to the bacterial flagellum, or to the main bacterial cell.

Explain why the antibodies are different, each one specific to its target.

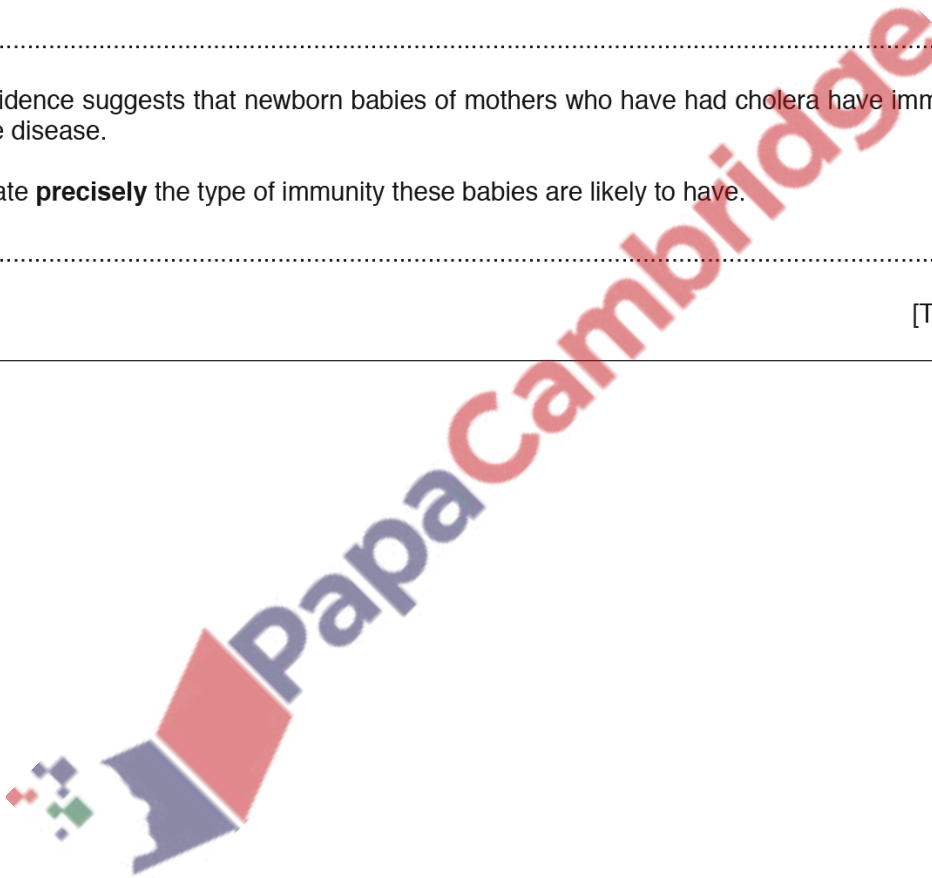
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- (h) Evidence suggests that newborn babies of mothers who have had cholera have immunity to the disease.

State **precisely** the type of immunity these babies are likely to have.

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[Total: 19]



206. 9700_w17_qp_23 Q: 3

Fig. 3.1 shows the structure of an alveolus and surrounding structures in a mammalian lung. The lining of each alveolus is formed by two types of epithelial cell, alveolar type 1 and alveolar type 2.

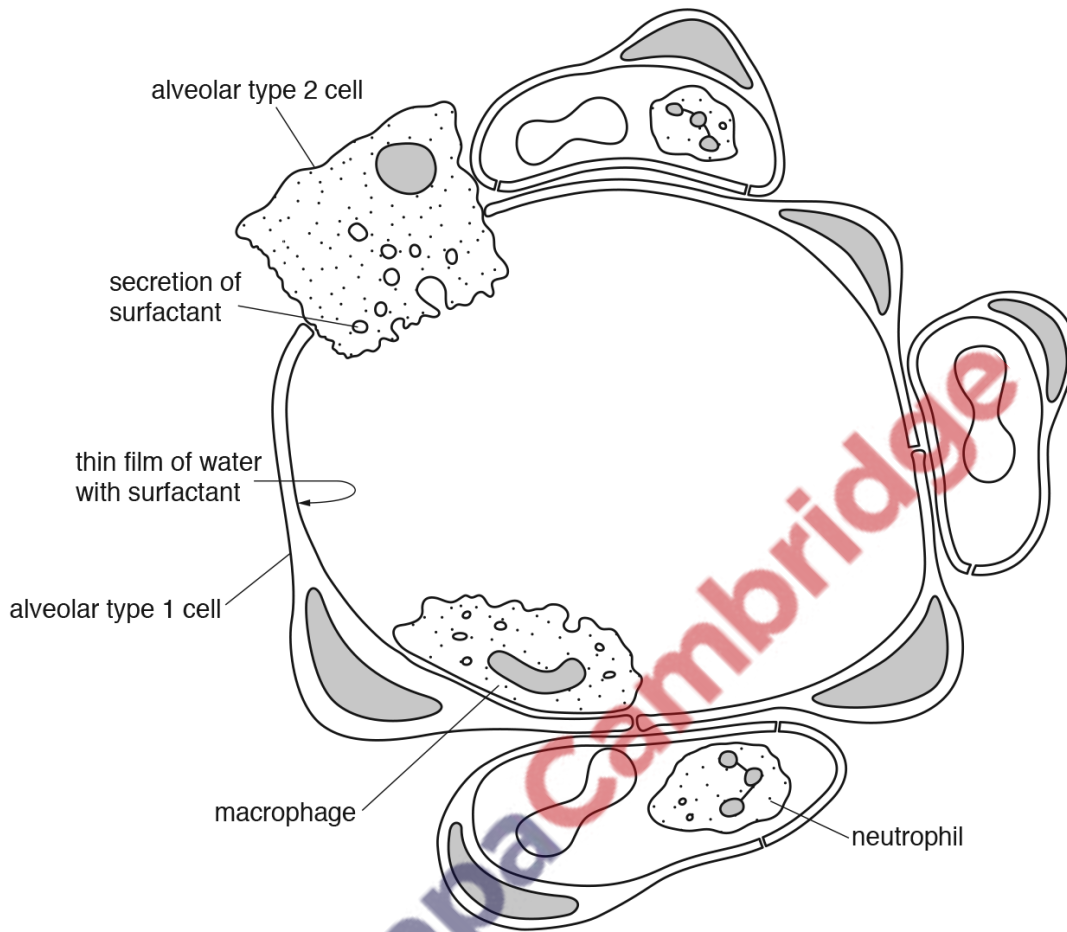


Fig. 3.1

not to scale

(a) Explain how the structure of an alveolar type 1 cell is adapted to its function.

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.....[2]

- (b) Alveolar type 2 cells secrete pulmonary surfactant into the watery fluid that lines the alveolus. The surfactant reduces the surface tension of the fluid so that the alveolus does not collapse.

Pulmonary surfactant is a mixture of phospholipids and proteins. The phospholipids form a monolayer on the surface of the fluid.

Explain how phospholipids interact with water to form a monolayer on the surface of the fluid.

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Macrophages and neutrophils are found in the lungs, as shown in Fig. 3.1.

- (c) Describe the role of macrophages in the lungs.

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- (d) Neutrophils leave the blood and secrete the extracellular enzyme, elastase.

- (i) Suggest why neutrophils secrete elastase.

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- (ii) The protein alpha-1 antitrypsin is produced in cells in the liver and is transported to the lungs, where it inhibits the action of elastase.

Some people produce a different form of this protein that remains within liver cells. These people are at an increased risk of developing emphysema, in which alveolar walls break down. Emphysema is one of the conditions associated with chronic obstructive pulmonary disease (COPD).

Explain why these people are at increased risk of developing emphysema.

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[3]

[Total: 12]

207. 9700_s16_qp_21 Q: 2

Macrophages synthesise intracellular enzymes.

Fig. 2.1 is a summary diagram of events that occur in a macrophage.

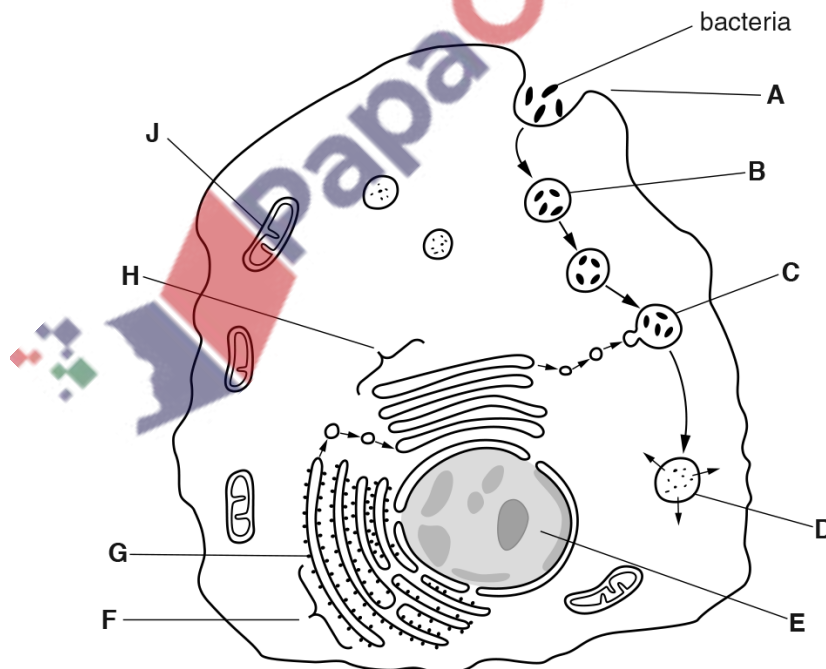


Fig. 2.1

(a) (i) Name the process at **A**.

.....[1]

(ii) Name the stages of protein synthesis that occur at **E** and at **F**.

E

F[2]

(iii) Name organelles **B**, **G**, **H** and **J**.

B

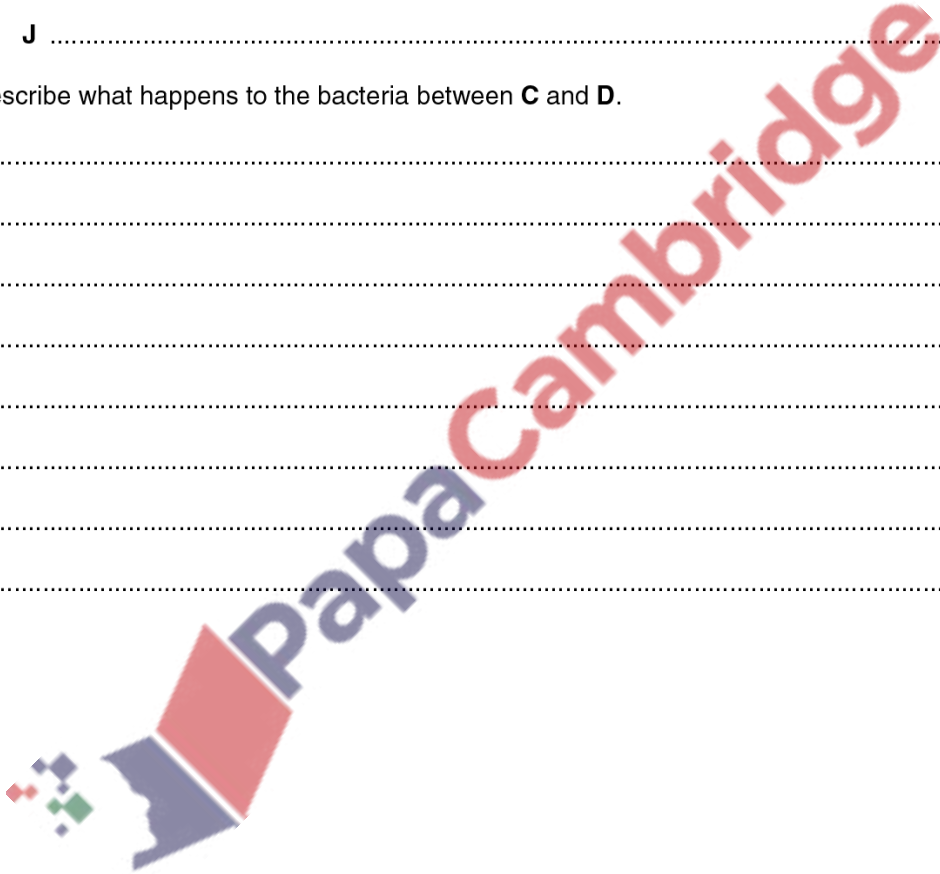
G

H

J[4]

(b) Describe what happens to the bacteria between **C** and **D**.

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.....[3]



- (c) Macrophages are antigen presenting cells (APCs). Antigens from pathogens such as the bacteria shown in Fig. 2.1 are presented to helper T-lymphocytes as shown in Fig. 2.2.

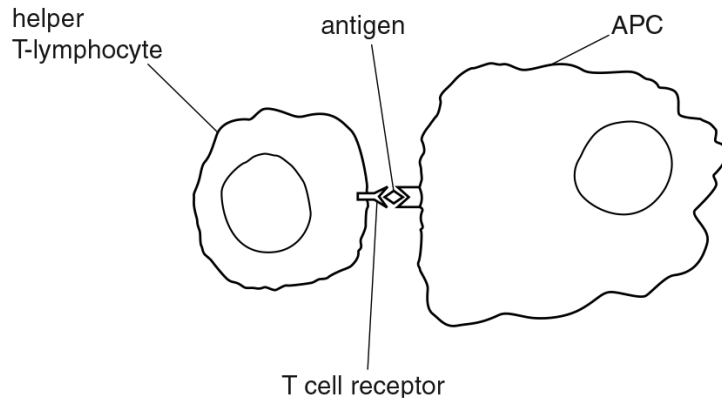


Fig. 2.2

Very few helper T-lymphocytes respond to the presence of APCs by binding in the way shown in Fig. 2.2.

Suggest why this is so.

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.....[2]

[Total: 12]



208. 9700_s15_qp_21 Q: 2

Pathogens enter the body in a variety of ways, including through the gas exchange system. The body has several defence mechanisms against the entry of pathogens and their spread throughout the body.

Fig. 2.1 is an electron micrograph of a cross section of the lining of a bronchiole.



Fig. 2.1

(a) (i) Name tissue X and cell Y.

X

Y[2]

(ii) With reference to the structures visible in Fig. 2.1, state three ways in which the lining of the trachea, bronchus and bronchioles provides protection against the entry of bacterial pathogens.

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.....[3]

Fig. 2.2 shows part of the immune response to the first infection by a bacterial pathogen that has entered the body through the lining of a bronchiole. **J** and **K** are stages in the immune response.

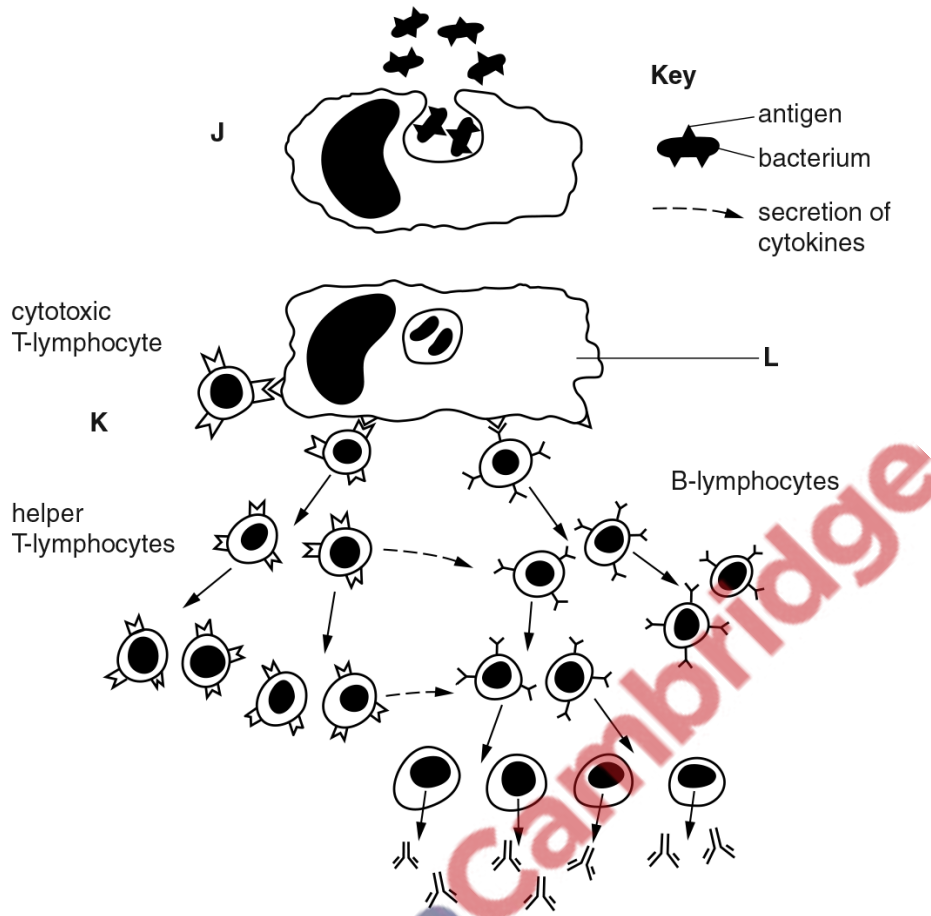


Fig. 2.2

(b) (i) State what is happening at stage **J**.

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[1]

(ii) Explain the role of cell **L** at stage **K** in the immune response.

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[2]

209. 9700_w15_qp_21 Q: 5

- (a) State the name of the causative organism (pathogen) of measles.

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- (b) Describe how the measles pathogen is transmitted.

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- (c) The measles pathogen contains a single stranded RNA molecule and no DNA.

The cells that the measles pathogen infects contain double stranded DNA molecules.

State two **other** ways in which the RNA in the measles pathogen differs from the DNA in the infected cells.

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2.
..... [2]

- (d) The measles pathogen must carry out RNA replication to make new RNA molecules for the new pathogens. This happens inside the infected cell.

The pathogen carries its own enzyme for RNA replication, but no other enzymes.

Explain why the measles pathogen cannot use an enzyme from the cell to carry out RNA replication.

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..... [2]

- (e) The outer part of the measles pathogen contains an antigen called the H-protein. This antigen appears on the surface of cells infected with the measles pathogen.

Describe the role played by T-lymphocytes in a primary immune response to an infection by the measles pathogen.

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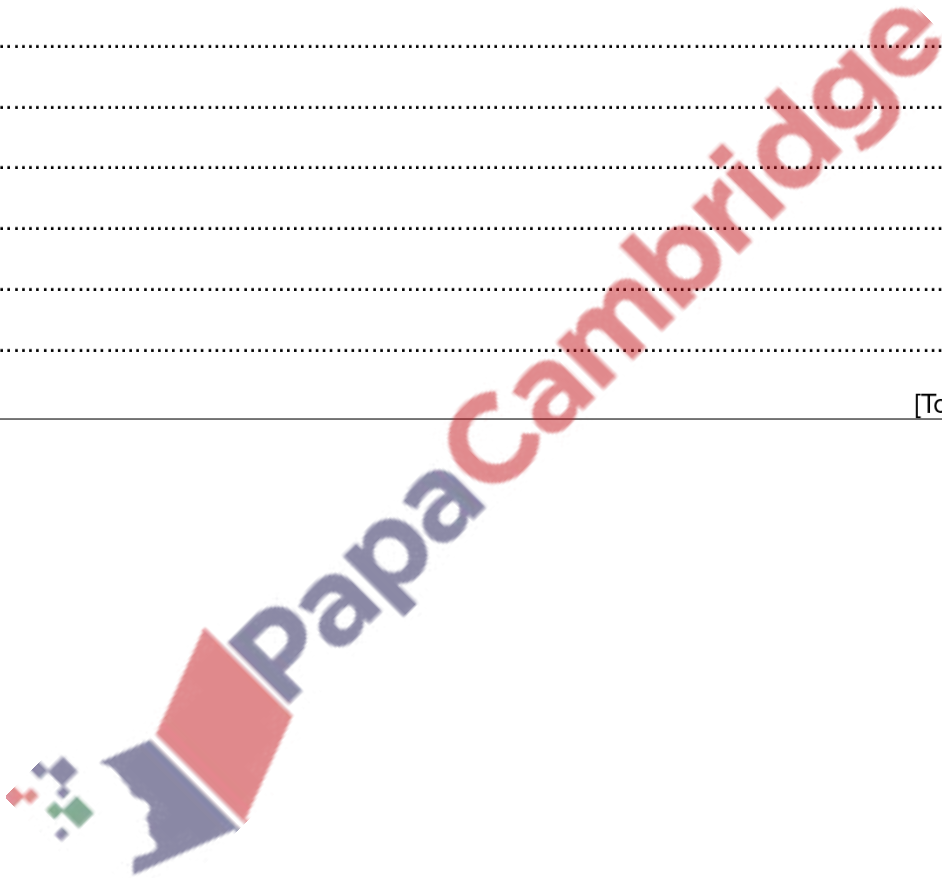
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[Total: 12]



210. 9700_w15_qp_22 Q: 2

Tobacco smoking is known to be associated with atherosclerosis and emphysema.

(a) Outline ways in which tobacco smoking can contribute to atherosclerosis.

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(b) Fig. 2.1 is a scan of the lungs of a person with emphysema. One common feature in the damaged areas labelled is a loss of the elastic fibres of the alveoli. Another feature is an increased number of macrophages and neutrophils.

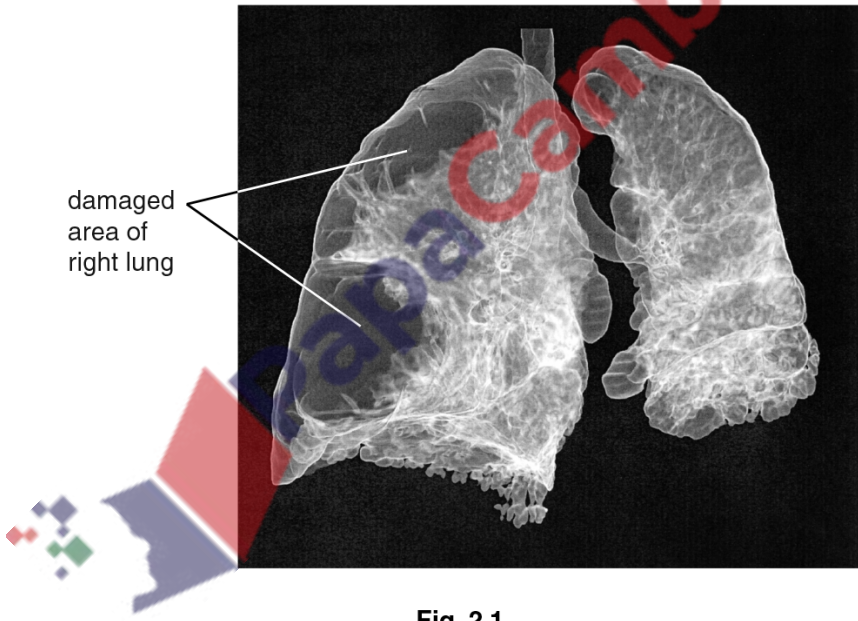


Fig. 2.1

(i) State the general role shared by macrophages and neutrophils.

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- (ii) Suggest how the loss of the elastic fibres would cause the enlargement of the lung shown in Fig. 2.1.

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..... [2]

- (c) The synthesis and release of elastase enzymes by macrophages and neutrophils is an important feature in the development and progression of emphysema. Elastase causes the breakdown of the protein elastin, the main component of elastic fibres.

- (i) Explain what is meant by an enzyme.

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..... [2]

- (ii) Elastase has an active site with a specific shape. The mode of action of this enzyme supports the lock and key hypothesis.

Explain the mode of action of elastase.

You may use the space below to draw a diagram or diagrams to help your answer.

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..... [3]

(d) There are two inhibitors of elastase that are produced in the body, TIMP-1 and A1AT:

- macrophage elastase is inhibited by TIMP-1
- neutrophil elastase is inhibited by A1AT.

The inhibitors can be inactivated by the elastase enzymes:

- macrophage elastase can inactivate A1AT
- neutrophil elastase can inactivate TIMP-1.

In healthy lungs, the activity of elastase enzymes is regulated. Tobacco smoke can disrupt this regulation.

(i) One effect of tobacco smoke is to cause changes in the structure of A1AT, a competitive inhibitor.

Suggest how structural changes to A1AT will affect its mode of action.

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..... [1]

(ii) A1AT is a protein. Some non-smokers have a mutation in the gene coding for A1AT and are at risk of developing emphysema as there is a lack of A1AT in the lung tissue.

Explain why a lack of A1AT in these non-smokers means that they are at risk of developing emphysema.

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211. 9700_w15_qp_22 Q: 4

(a) Table 4.1 describes three examples of substances moving into or out of cells.

Complete Table 4.1 by identifying the transport mechanism involved for each example.

Table 4.1

example	transport mechanism involved
uptake of magnesium ions from a lower concentration in the soil solution to a higher concentration in the cytoplasm of a root hair cell	
release of antibodies from an active B-lymphocyte (plasma cell)	
movement of sucrose from a companion cell into a phloem sieve tube element via plasmodesmata	

[3]

(b) Oxygen moves into and out of red blood cells. Fig. 4.1 shows an oxygen dissociation curve for adult human haemoglobin.

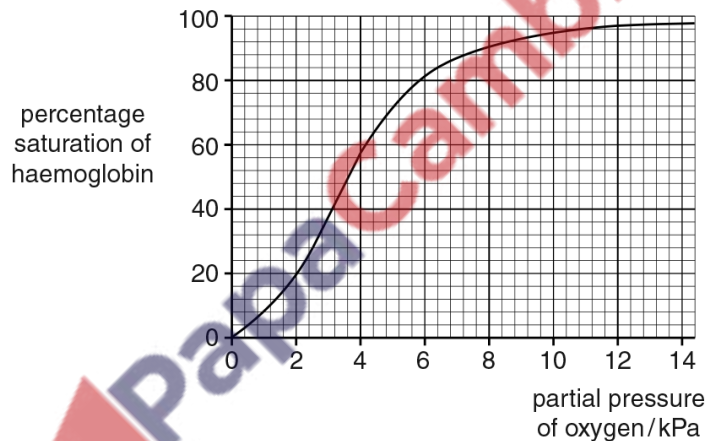


Fig. 4.1

◆ The steepest part of the curve is between 2.6 kPa and 4.2 kPa.

Explain the importance of this for respiring tissues.

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[Total: 5]

11.2 Antibodies and vaccination

212. 9700_m20_qp_22 Q: 5

Myasthenia gravis and HIV/AIDS both involve disorders of the immune system.

(a) Outline why myasthenia gravis is described as a disorder of the immune system.

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A person with HIV/AIDS has a weakened immune system. This is because HIV infects cells of the immune system, in particular T-helper lymphocytes (T_h cells). The pathogen can remain inactive within host cells. In some people, the pathogen becomes active and causes the number of T_h cells to decrease.

Antiretroviral therapy (ART) is used to treat people who are infected with HIV (living with HIV). ART aims to keep the number of T_h cells at a healthy level.

(b) State the full name of the pathogen known as HIV.

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(c) Explain why it is important that ART maintains a healthy number of T_h cells in a person living with HIV.

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(d) Fig. 5.1 shows global estimates of:

- the percentage of people living with HIV who received treatment with ART in each year from 2000 to 2015
- the number of people who died from HIV/AIDS in each year from 2000 to 2015.

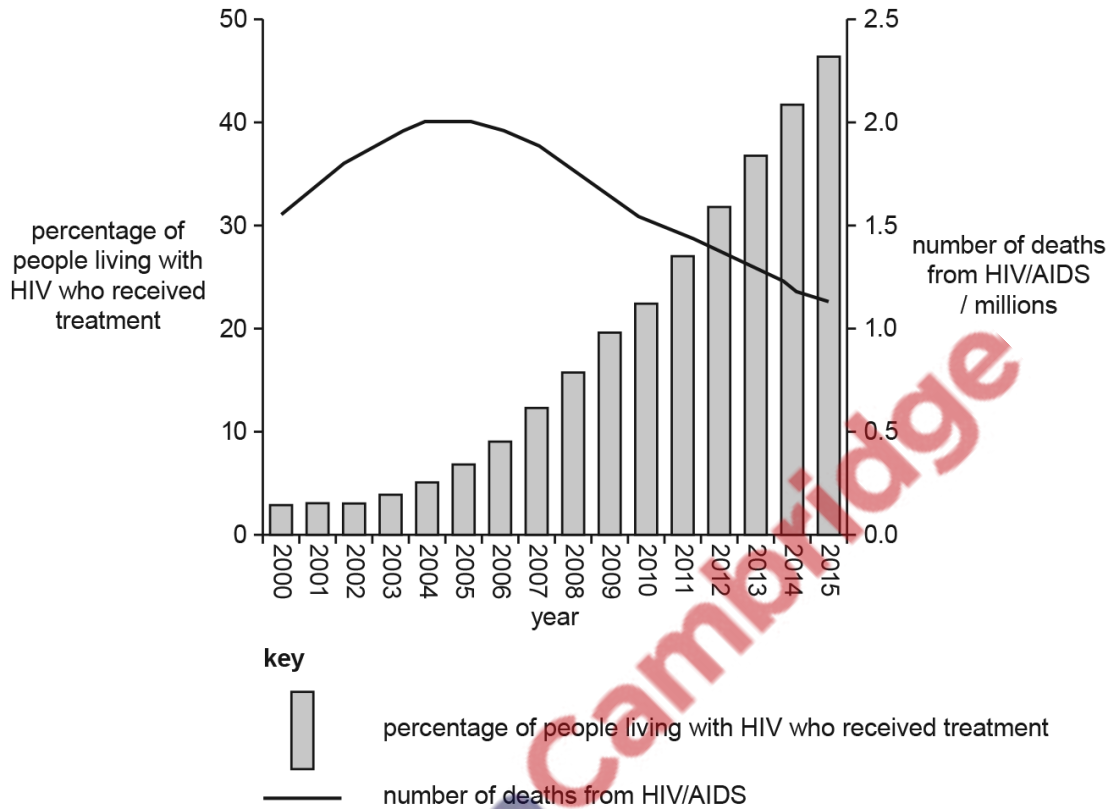


Fig. 5.1

(i) Describe the trends shown in Fig. 5.1.

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[3]

(ii) It is recommended that ART is given to all people living with HIV.

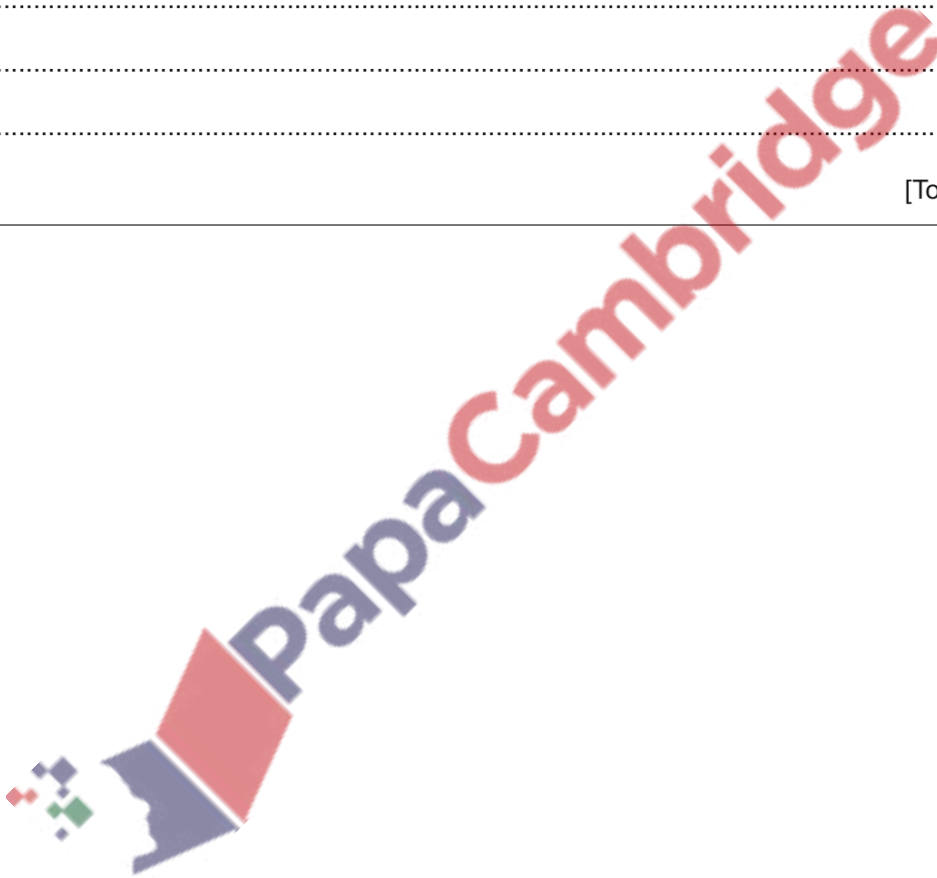
Some countries that support this recommendation find it difficult to provide ART to everyone living with HIV.

Other than the high cost of treatment, suggest **two** reasons why it is difficult to provide ART to everyone living with HIV.

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[2]

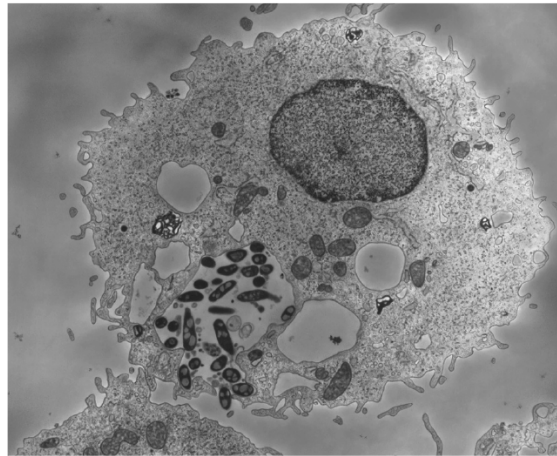
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213. 9700_s20_qp_21 Q: 5

The vaccine used to control tuberculosis (TB) is known as Bacillus Calmette-Guérin (BCG). The vaccine contains live bacteria that have been selected so that they do not cause disease in humans.

Fig. 5.1 shows a macrophage that is in the process of engulfing the bacteria in the vaccine.



magnification $\times 4400$

Fig. 5.1

(a) (i) Name the pathogen that causes TB.

..... [1]

(ii) Describe how this pathogen is transmitted.

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- (b) Describe the events that occur in the body after the macrophage has engulfed the bacteria until the production of antibodies in response to the BCG vaccine.

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- (c) Vaccines, such as BCG, stimulate the formation of memory cells.
Explain the role of memory cells in the body's defence against the TB pathogen.

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(d) Suggest why vaccination with BCG has not yet eradicated TB.

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[Total: 14]

PapaCambridge

214. 9700_s20_qp_22 Q: 3

The Bacillus Calmette-Guérin (BCG) vaccine is the only vaccine used to provide protection against the infectious bacterial disease tuberculosis (TB). Most countries of the world have a BCG vaccination programme.

- (a) TB is most commonly transmitted from person to person by aerosol infection. The causative organism is present in airborne droplets.

Name the species of causative organism of TB commonly passed from person to person by aerosol infection.

..... [1]

- (b) In general, the countries that do not have a BCG vaccination programme are high-income countries that have a low number of cases of TB. In most of these countries, the vaccine is given only to babies and children at high risk of developing TB.

Suggest **one** reason why a child in a country with a low number of cases of the disease could be at a high risk of developing TB.

.....

 [1]

- (c) Countries are classified by the World Bank into one of four income groups.

Table 3.1 shows the estimated incidence of TB for 2012 to 2016 for these income groups.

The incidence represents the number of new cases of TB occurring per 100 000 people in one year. The new cases include the number of cases that have occurred again after a period of recovery (relapse TB).

Table 3.1

		incidence per 100 000 people				
income group	year	2012	2013	2014	2015	2016
	low		253	244	238	231
lower middle		244	240	236	232	227
upper middle		84	81	78	76	74
high		14	13	13	12	12

Describe the patterns and trends shown in Table 3.1.

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- (d) There is evidence that the BCG vaccine has also provided protection against the disease leprosy.

Leprosy is caused by a bacterium that is closely related to the bacteria that cause TB.

Suggest why the BCG vaccine can also provide protection against leprosy.

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..... [2]

- (e) A baby can gain artificial active immunity to TB after having the BCG vaccine. A baby can also gain natural passive immunity to TB.

State the differences between **artificial active** immunity and **natural passive** immunity.

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[Total: 10]

215. 9700_s20_qp_23 Q: 2

In 2016, the highest number of cases of malaria and deaths caused by the disease were in sub-Saharan Africa. In many areas of sub-Saharan Africa, malaria is endemic (continually present) and people are at high risk of becoming infected with the *Plasmodium* pathogen.

In high risk areas it is recommended that:

- homes are provided with insecticide-treated nets (ITN)
- the surfaces inside homes where *Anopheles* mosquitoes may rest are sprayed with insecticide. This is known as indoor residual spraying (IRS).

(a) Explain how the use of ITN and IRS can help break the transmission cycle of malaria.

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..... [3]

(b) Fig. 2.1 shows the proportion of the population in sub-Saharan Africa at risk of malaria that is protected by using IRS or ITN, or both, in the years 2010 to 2016.

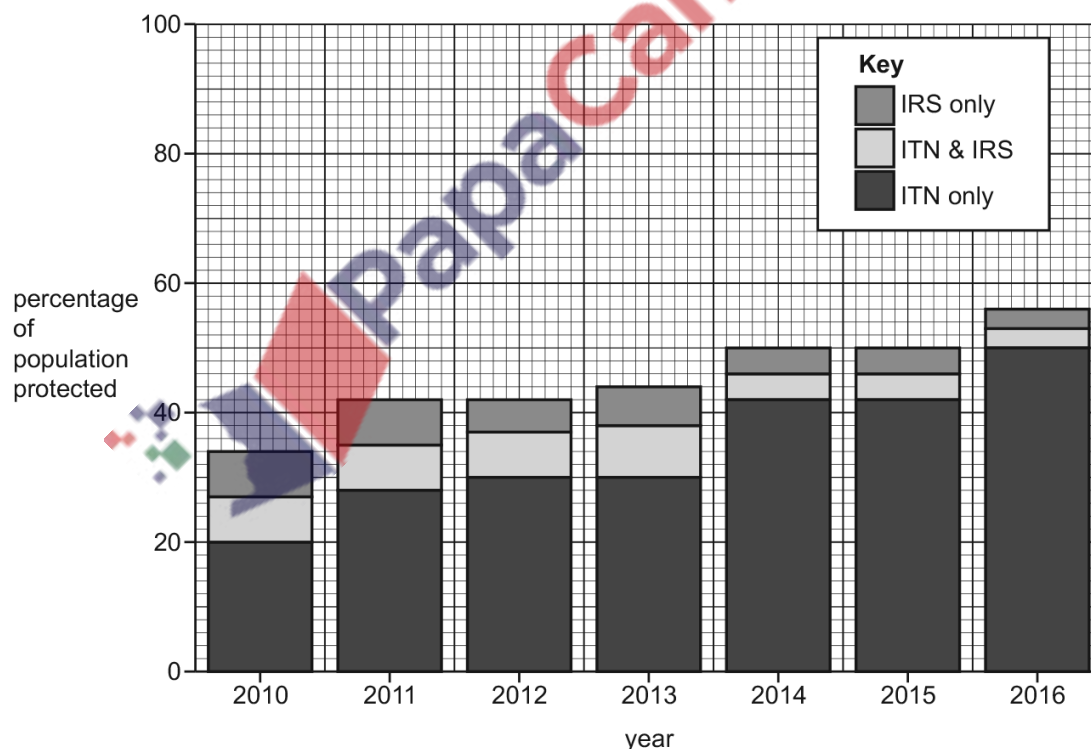


Fig. 2.1

The main trend in Fig. 2.1 shows that there is an increase in the percentage of the population protected over time.

- (i) State **one** other trend shown in Fig. 2.1.

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..... [1]

- (ii) Explain why the main trend shown in Fig. 2.1 could be a concern for the World Health Organization.

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..... [1]

- (iii) With reference to Fig. 2.1, suggest a reason for the difference in trends shown for ITN only compared with IRS only.

.....
..... [1]

- (c) In a primary immune response, antibodies against *Plasmodium* are produced within one to two weeks following infection. In some people, the pathogen is eliminated and the concentration of antibodies in the circulation decreases over time.

Infection again by *Plasmodium* with the same antigens causes a secondary response that also involves antibody production.

State **and** explain how the antibody response following a second infection will differ from the primary immune response.

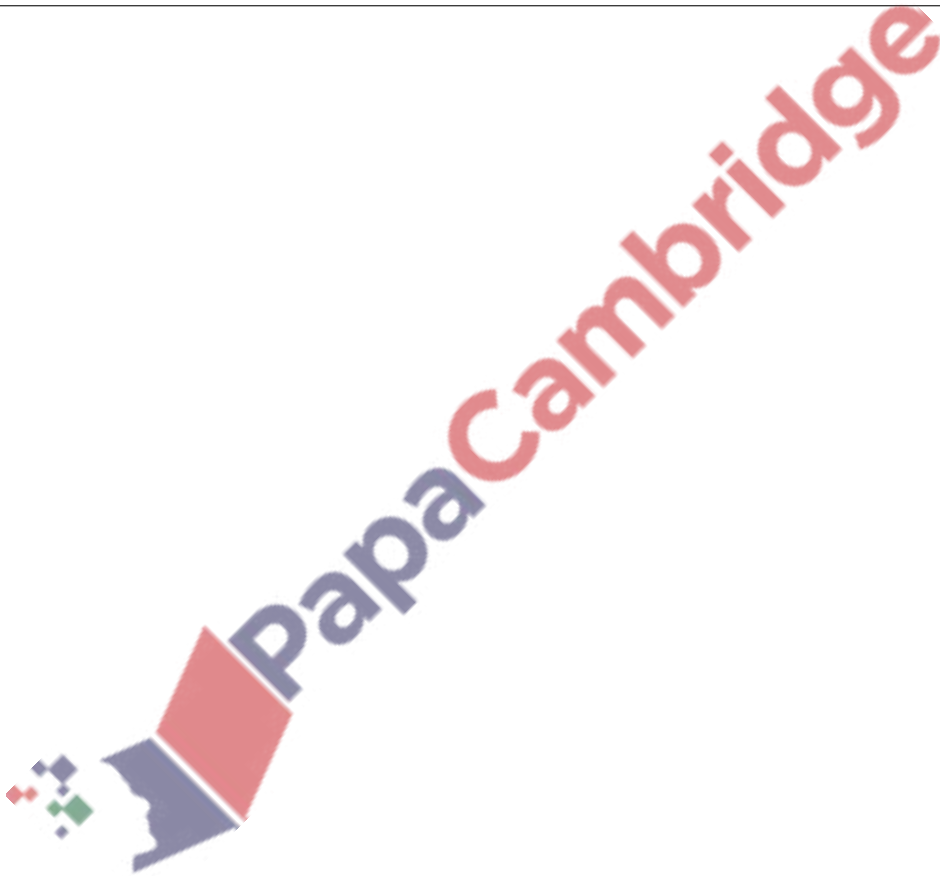
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- (d) In malaria, the production of antibodies is beneficial to recovery, whereas in the disease myasthenia gravis the production of antibodies is harmful.

Explain why the production of antibodies in a person with myasthenia gravis is harmful.

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..... [2]

[Total: 11]



216. 9700_w20_qp_22 Q: 4

In the immune system, a plasma cell develops from an activated B-lymphocyte. Mature plasma cells synthesise and secrete antibody molecules.

(a) Fig. 4.1 is a diagram of a transmission electron micrograph of a plasma cell.

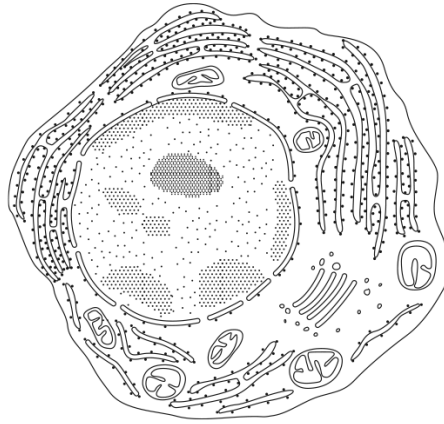


Fig. 4.1

The plasma cell can be seen in greater detail using an electron microscope compared with using a light microscope.

(i) Describe the **extra** detail of the nucleus that can be seen using an electron microscope.

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..... [3]

(ii) Explain why cell structures, such as ribosomes and the rough and smooth endoplasmic reticulum, cannot be seen using a light microscope.

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..... [2]

- (b) The transition from the activated B-lymphocyte to the fully mature plasma cell requires a number of mitotic cell cycles to occur. This process, which is known as clonal expansion, results in a large number of genetically identical plasma cells.

Fig. 4.2 describes events, **A** to **F**, that occur during the mitotic cell cycle of the B-lymphocyte.

A centrioles replicate
B DNA polymerase catalyses the formation of phosphodiester bonds
C condensation of chromosomes
D nuclear envelope reassembles around each set of daughter chromosomes
E centromeres move towards poles
F chromosomes line up at spindle equator

Fig. 4.2

Table 4.1 lists the stages occurring during one cell cycle of the B-lymphocyte. These stages are not in the correct order.

Table 4.1

stage of cell cycle	correct letter from Fig. 4.2
G ₂ phase	
metaphase	F
cytokinesis	
prophase	
S phase	
anaphase	
G ₁ phase	
telophase	

Complete Table 4.1 by writing the letter of the event described in Fig. 4.2 that correctly matches the stage of the cell cycle listed.

Leave a **blank space** if there is **no** matching description for the stage in the list. Use each letter **once** only.

One of the letters in Table 4.1 has already been added for you.

[5]

- (c) Clonal expansion also results in the production of memory B-lymphocytes.

Explain the importance of clonal expansion **and** the production of memory B-lymphocytes in providing protection for a person against an infectious disease.

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- (d) Myasthenia gravis is an example of a disease where the immune system fails to distinguish between self and non-self.

Explain what is meant by this statement.

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..... [2]

[Total: 15]



217. 9700_m19_qp_22 Q: 3

In 2015, the World Health Organization (WHO) published the Global Technical Strategy for Malaria 2016–2030. The aim of this global strategy, which follows on from the 2008 Global Malaria Action Plan (GMAP), is to make progress in the control and elimination of malaria.

Both the global strategy and GMAP aim to reduce:

- the case incidence (number of new cases each year) of malaria
- the mortality rate (number of deaths each year) from malaria.

(a) Fig. 3.1 shows data for the four countries in the WHO Western Pacific Region that had the highest proportion of cases of malaria in 2015.

For each of these four countries, the percentage change in the case incidence and the percentage change in the mortality rate over the five-year period from 2010 to 2015 are shown.

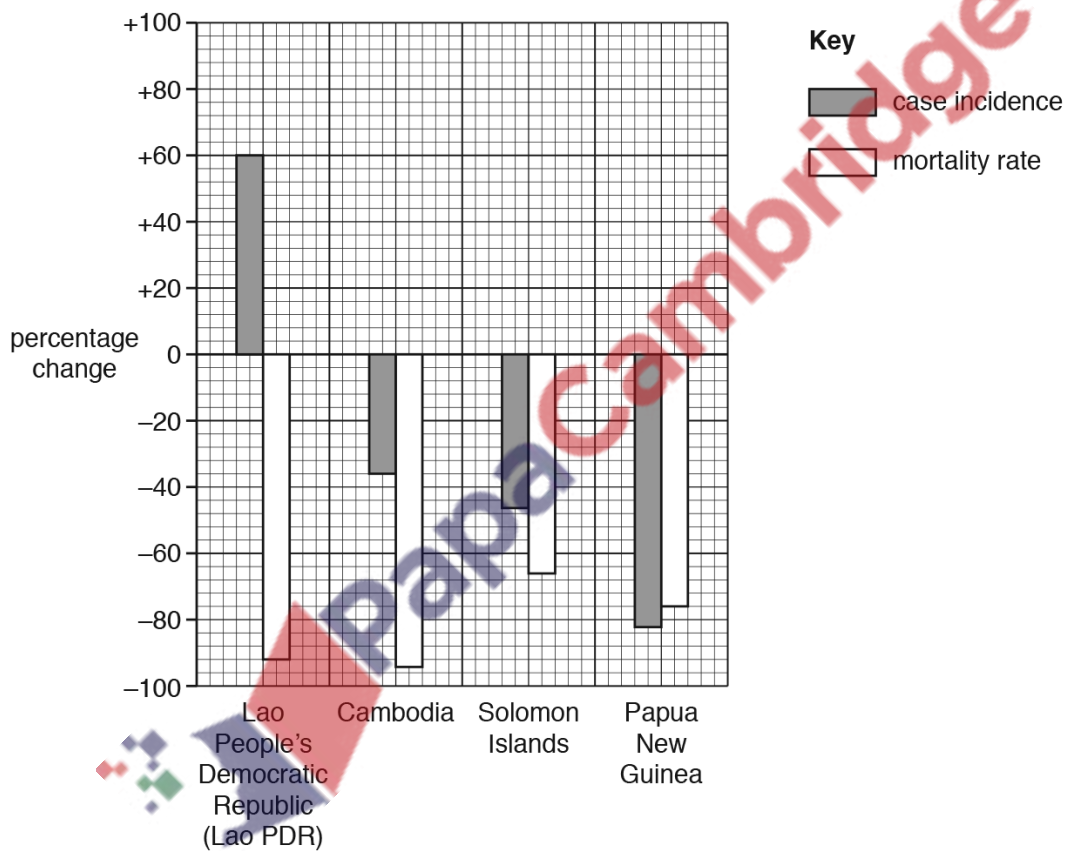


Fig. 3.1

- (i) With reference to Fig. 3.1, describe the progress made in the control of malaria in the four countries between 2010 and 2015.

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- (ii) All the countries shown in Fig. 3.1 supplied households at risk of malaria with insecticide-treated nets (ITNs). This is one of the recommendations in the GMAP and the global strategy.

Describe **and** explain the role of ITNs.

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- (b) Another recommendation of the global strategy is to carry out rapid diagnostic testing (RDT) of individuals who may have malaria. This involves testing human blood samples for the presence of proteins specific to *Plasmodium*. Test sticks can be used.

Table 3.1 contains information about two RDT test sticks.

Table 3.1

test stick	<i>Plasmodium</i> protein tested for	species of <i>Plasmodium</i> that produce the protein
1	pLDH (parasite lactate dehydrogenase)	<i>P. vivax</i> <i>P. falciparum</i> <i>P. ovale</i> <i>P. malariae</i>
2	HRP-2 (histidine-rich protein 2)	<i>P. falciparum</i> only

Some details of the design of these RDT test sticks are shown in Fig. 3.2.

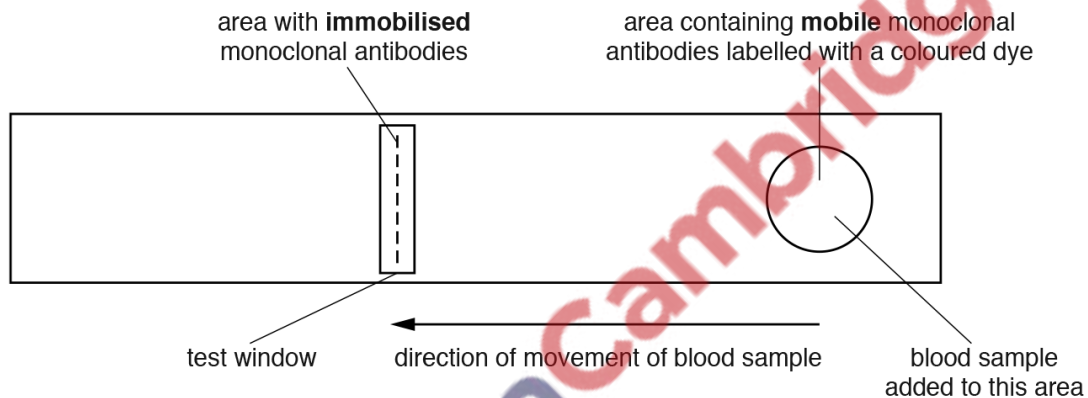


Fig. 3.2

The **immobilised** monoclonal antibodies in the test window are not visible.

If the blood sample contains a *Plasmodium* protein that can be detected by the RDT test stick:

- the **mobile** monoclonal antibodies bind to one part of the protein
- the **immobilised** monoclonal antibodies bind to another part of the protein
- a coloured line in the test window indicates a positive result for the protein.

- (i) With reference to Table 3.1 and Fig. 3.2, explain why test stick 1 and test stick 2 will contain **different** mobile monoclonal antibodies.

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..... [2]

- (ii) Two blood samples were removed from a person. One sample was added to test stick 1 and the other sample was added to test stick 2.

With reference to Table 3.1 and Fig. 3.2, explain what can be diagnosed for this person from a **positive** result for test stick 1 and a **negative** result for test stick 2.

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..... [2]

[Total: 10]



218. 9700_s19_qp_21 Q: 1

- (a) Antibody molecules are proteins that show primary structure, secondary structure, tertiary structure and quaternary structure.

Fig. 1.1 shows a ribbon diagram of an antibody molecule.

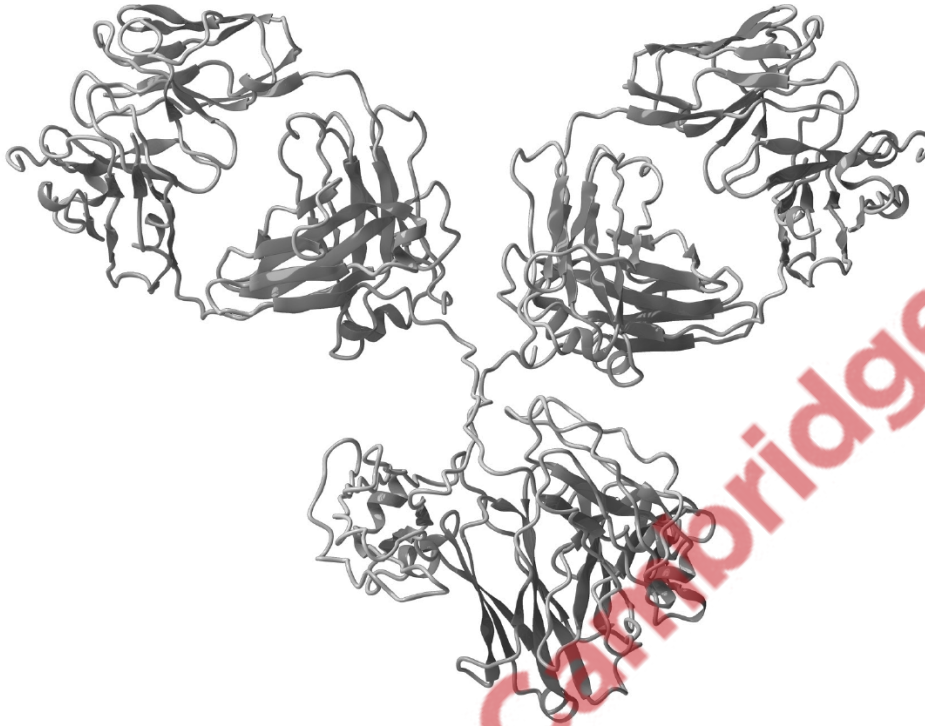


Fig. 1.1

Describe how Fig. 1.1 shows the secondary structure **and** tertiary structure of the antibody molecule.

secondary structure

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tertiary structure

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[3]

(b) Fig. 1.2 is a transmission electron micrograph of a hybridoma cell.

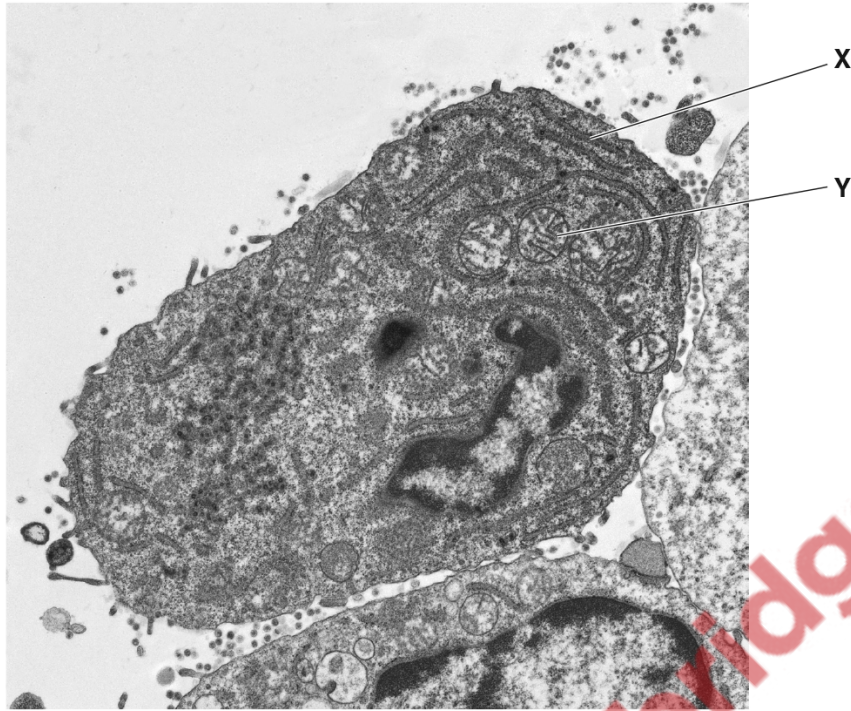


Fig. 1.2

- (i) The hybridoma cell in Fig. 1.2 synthesises and secretes molecules of a monoclonal antibody.

State the roles of the structures labelled X and Y in the production of antibody molecules in the hybridoma cell.

X

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.....

Y

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[2]

- (ii) The hybridoma method for the production of monoclonal antibodies involves a number of stages. One of these stages is the formation of hybridoma cells.

Outline the stage in which hybridoma cells are formed.

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- (iii) Outline the use of monoclonal antibodies in the treatment of disease.

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..... [3]

[Total: 11]

219. 9700_w19_qp_21 Q: 2

Fig. 2.1 is a transmission electron micrograph showing the bacterial pathogen that causes tuberculosis (TB).

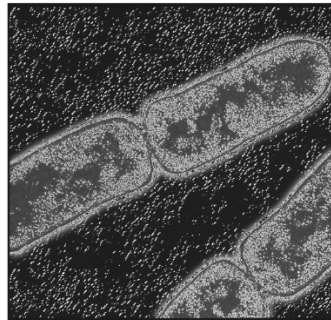


Fig. 2.1

(a) (i) Name the pathogen shown in Fig. 2.1 that causes TB.

..... [1]

The World Health Organization (WHO) introduced a strategy in 2015 to end the global TB epidemic.

An important part of the strategy is to:

- identify people at risk of becoming infected with TB
- use methods to prevent transmission of TB.

The BCG vaccination is one method of prevention recommended for use in countries where TB is common. The BCG vaccine contains a non-pathogenic, living form of the microorganism that causes TB.

(ii) Complete Table 2.1 by using a tick (✓) to identify the type of immunity that develops in a person who has been given the BCG vaccination.

Table 2.1

artificial active immunity	
artificial passive immunity	
natural active immunity	
natural passive immunity	

[1]

(b) Rifampicin is one of the antibiotics used to treat TB.

Rifampicin inhibits RNA polymerase in bacterial cells by binding to a site other than the active site. This prevents polypeptide synthesis.

(i) Suggest **and** explain how rifampicin prevents polypeptide synthesis in bacterial cells.

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..... [3]

Some bacteria have developed resistance to rifampicin. However, they are still susceptible to the other antibiotics that can be used to treat TB.

Multi-drug resistant bacteria have developed resistance to at least two drugs, including rifampicin.

WHO collects data from all countries on the number of cases of TB caused by rifampicin-resistant bacteria (RR-TB) and multi-drug resistant bacteria (MDR-TB).

Fig. 2.2 shows the reported number of cases of TB between 2009 and 2013.

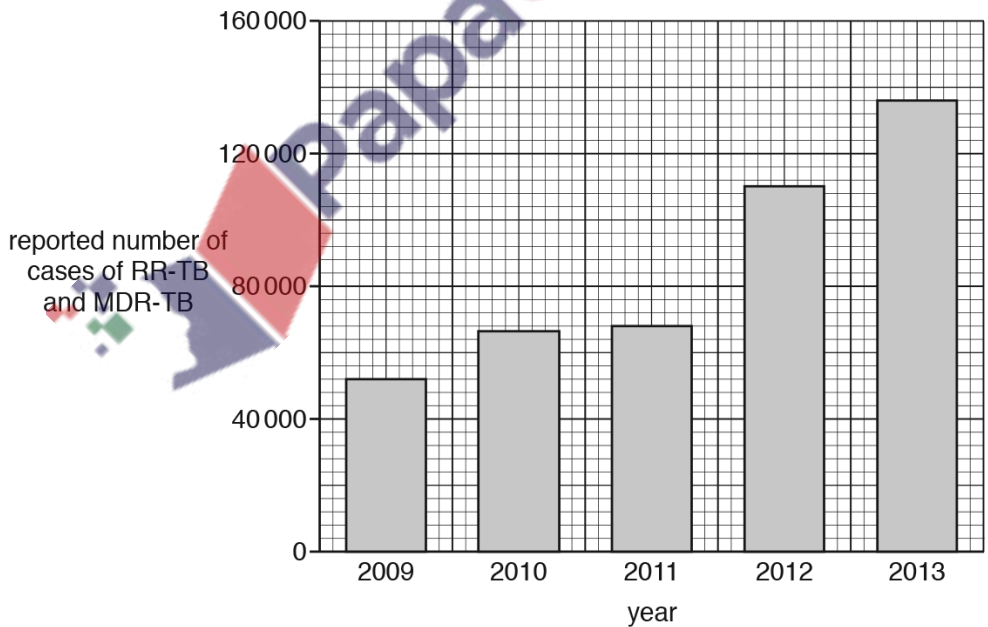


Fig. 2.2

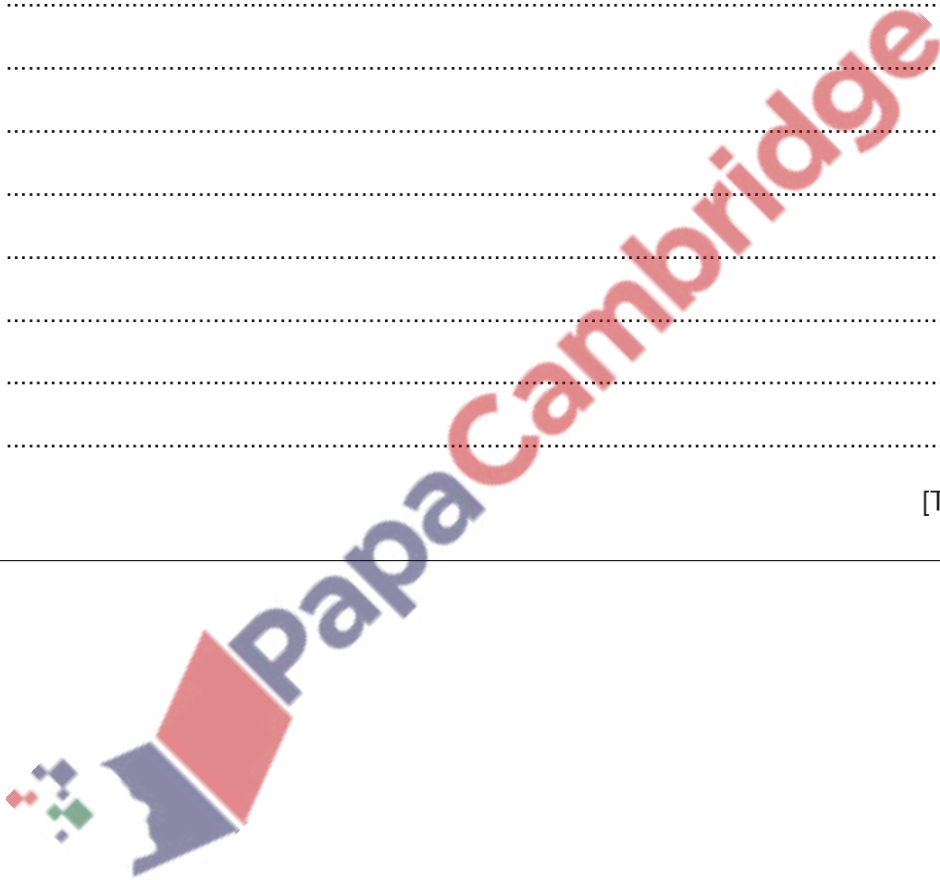
(ii) Describe the trend shown by the data in Fig. 2.2.

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..... [2]

(iii) Explain how resistance to drugs such as rifampicin develops.

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..... [4]

[Total: 11]



220. 9700_w19_qp_21 Q: 4

(a) Fig. 4.1 is a photomicrograph of a human blood smear.

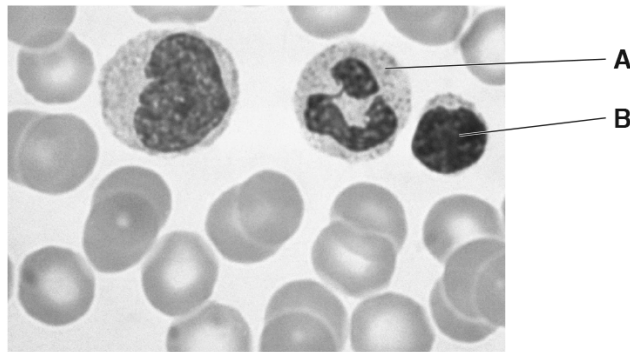


Fig. 4.1

Name the cells labelled **A** and **B** in Fig. 4.1.

A

B

[2]

(b) Blood and lymph are both fluids that transport substances within the human body.

(i) Table 4.1 shows components found in both blood and lymph.

Complete Table 4.1 to show whether the concentration of each of these components is **higher** or **lower** or the **same** in the lymph, when compared with the concentration in the blood in the aorta.

You may use the words **higher** or **lower** or **same** once, more than once or not at all.

Table 4.1

component	concentration in lymph compared to the concentration in blood in the aorta
oxygen	
carbon dioxide	
red blood cells	

[2]

- (ii) The presence of a pathogen infecting the body leads to an increase in the concentration of protein in lymph.

Suggest an explanation for this increase in protein concentration.

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..... [2]

- (c) Blood is circulated around the body by the heart.

The action of the heart is coordinated and controlled by structures located in its walls, such as the sinoatrial node (SAN) and the atrioventricular node (AVN).

- (i) Describe the role of the SAN.

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..... [2]

- (ii) In a healthy heart, the AVN provides the only pathway for electrical impulses to travel from the atria to the ventricles.

The bundle of Kent is a structure found in the heart in a small number of people.

Some electrical impulses do not pass through the AVN but travel directly from the atria to the ventricles through the bundle of Kent.

Suggest **and** explain the effects that the presence of the bundle of Kent may have on heart rate.

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..... [3]

[Total: 11]

221. 9700_w19_qp_21 Q: 5

Myasthenia gravis is a condition that results in muscle weakness by affecting the immune response.

(a) Explain what is meant by the term *immune response*.

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..... [2]


(b) Myasthenia gravis is an autoimmune disease which disrupts a cell signalling pathway involving muscle cells.

(i) Suggest how the immune system acts to disrupt this cell signalling pathway.

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..... [3]

Enzyme **Y** is found in the cell surface membrane of muscle cells. Enzyme **Y** acts to break down the cell signalling molecules which trigger muscle contraction when they are no longer needed.

(ii) Using the *induced fit* hypothesis of enzyme action, explain how enzyme **Y** breaks down the cell signalling molecules.



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..... [4]

- (c) Rituximab is a monoclonal antibody used in the treatment of myasthenia gravis.

Rituximab acts against the cell surface membrane protein CD20. This protein is found on the surface of the B-lymphocytes that cause myasthenia gravis.

Explain the advantages of using monoclonal antibodies in the treatment of diseases such as myasthenia gravis.

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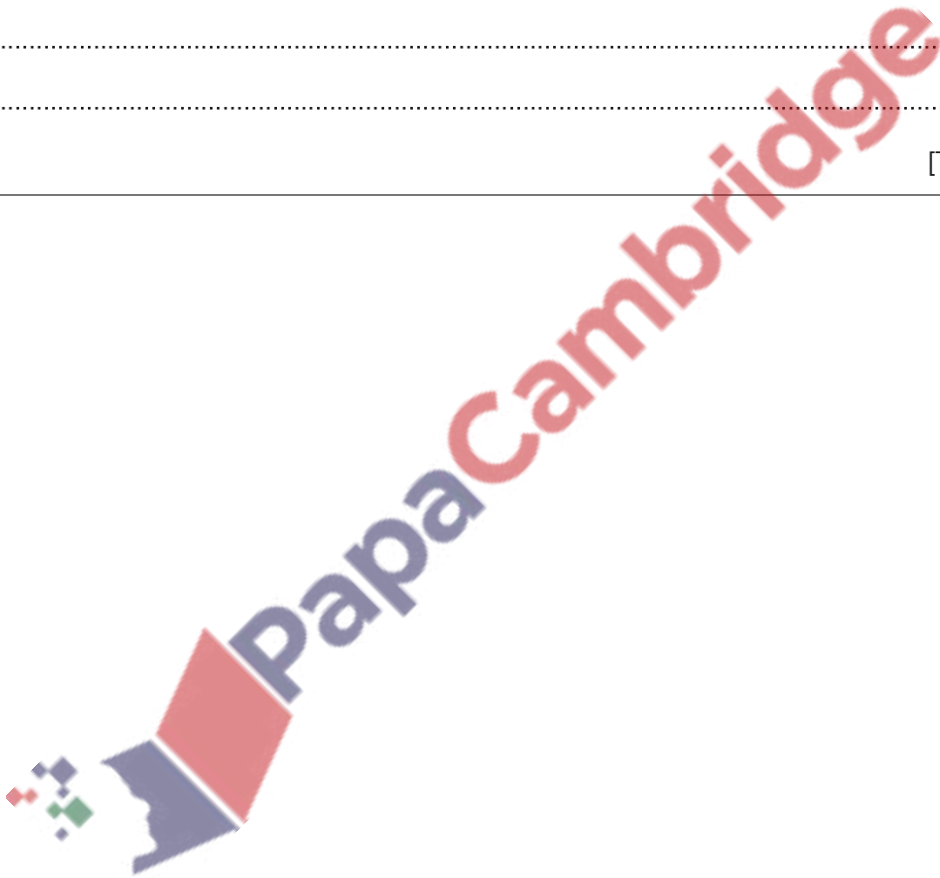
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[Total: 12]



222. 9700_w19_qp_22 Q: 5

Countries that have a high number of cases of malaria also have problems with diseases caused by bacteria. This means that many people in these countries are prescribed antibiotics, such as penicillin, for the treatment of bacterial infections.

(a) Outline how penicillin acts on bacterial cells.

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..... [3]

The female *Anopheles* mosquito is the vector of the *Plasmodium* pathogen that causes malaria. The insect takes in *Plasmodium* when feeding on blood from an infected person. At a later stage the insect can transmit the pathogen when taking a blood meal from an uninfected person.

(b) Name **one** of the four species of *Plasmodium* that can cause malaria.

..... [1]

(c) The male *Anopheles* mosquito does not feed on blood and so does not act as a vector.

Suggest why there is a difference in this feeding behaviour between male and female *Anopheles*.

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..... [1]

(d) Research has shown that *Plasmodium* is not always transmitted to uninfected people.

Two main reasons for this have been suggested.

- The immune system of mosquitoes kills *Plasmodium* while it is still in the gut.
- Bacteria living in the gut of mosquitoes compete with *Plasmodium* so it does not survive to continue its life cycle.

A study was carried out to see if taking antibiotics affects the risk of transmission of malaria.

Some observations and results of the study are summarised in Fig. 5.1.

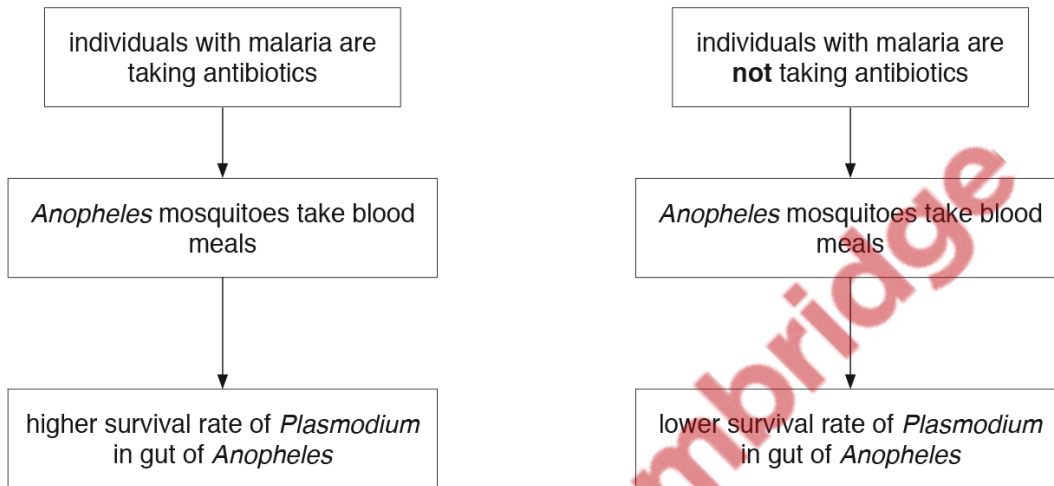


Fig. 5.1

Suggest explanations for the results shown in Fig. 5.1 **and** comment on the importance of these results for doctors working in countries that have malaria and a high number of bacterial infections.

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..... [3]

- (e) After many years of intense research and development, WHO reported in 2016 that a pilot vaccination programme would be trialled between 2017 and 2020.

The programme uses a vaccine acting against the most widespread species of *Plasmodium*.

Explain the difficulties faced by researchers in developing a malaria vaccine.

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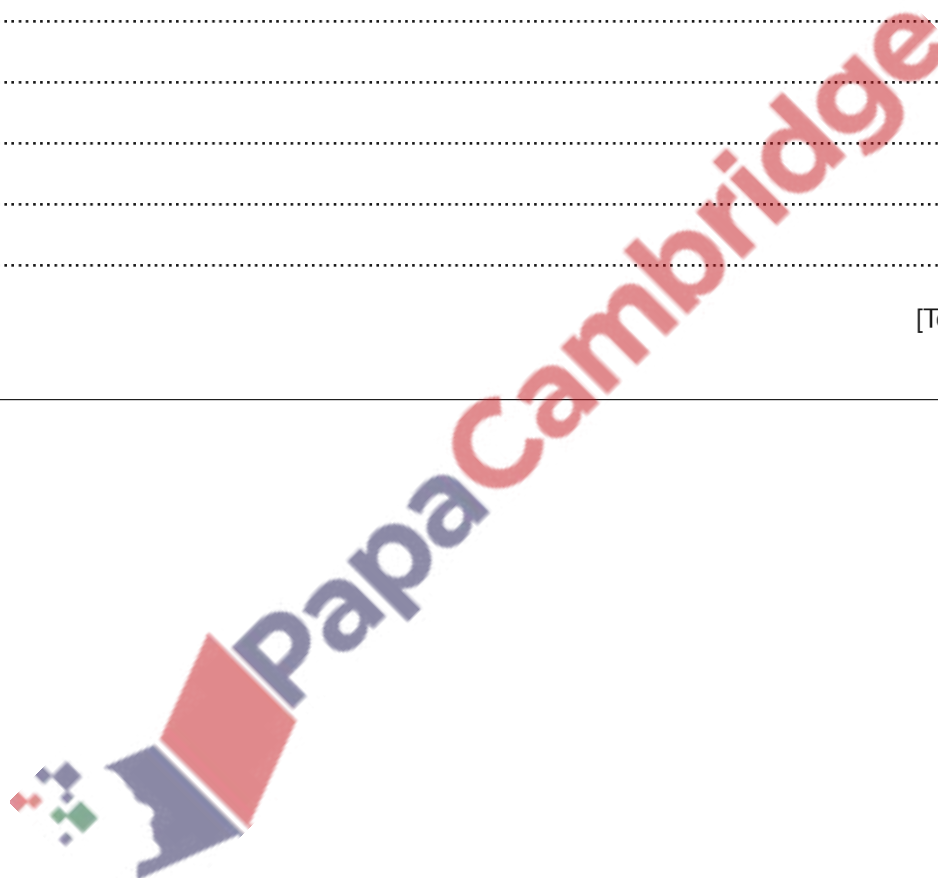
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..... [4]

[Total: 12]



223. 9700_w19_qp_23 Q: 5

Influenza is an infectious disease caused by the influenza A virus. This virus causes influenza in birds and mammals.

Fig. 5.1 is a diagram of an influenza A virus.

Haemagglutinin allows the virus to attach to host cells by binding to receptors on the cell surface membrane of the host cells.

Neuraminidase is an enzyme that helps the virus to leave host cells after the virus has replicated.

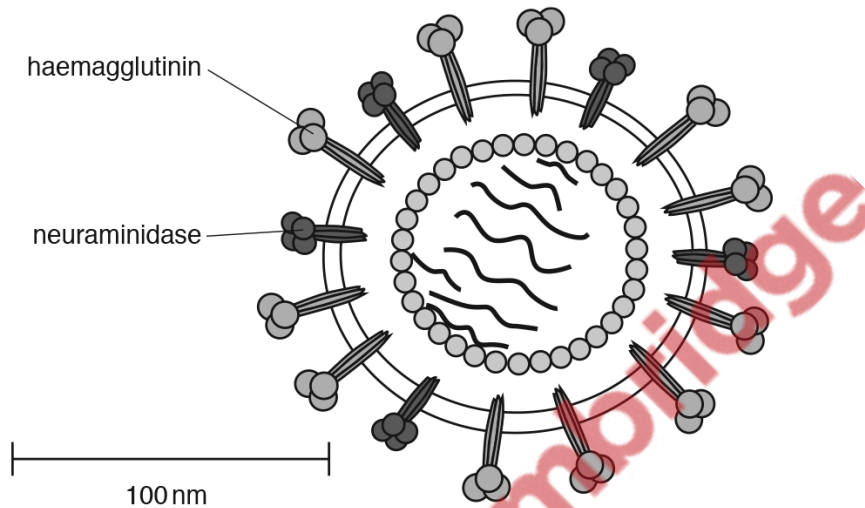


Fig. 5.1

(a) State **two** features of all viruses that are visible in Fig. 5.1.

- 1
- 2
- [2]

(b) Neuraminidase **removes** parts of the host cell receptors that bind to haemagglutinin. This helps newly-formed **viruses** to leave host cells.

Drugs have been **developed** to act on neuraminidase. These drugs prevent viruses from **leaving** host cells.

Suggest **and** explain how these drugs act to prevent viruses leaving cells.

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- [3]

- (c) The human immune system produces antibodies in response to the presence of antigens, such as neuraminidase and haemagglutinin.

Outline the events that occur during an immune response leading to the production of antibodies against an antigen, such as haemagglutinin.

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..... [4]

- (d) Researchers are developing methods to produce antibodies to give artificial passive immunity to influenza.

- (i) Suggest the advantages **and** disadvantages of artificial passive immunity.

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..... [3]

- (ii) State **two** ways in which mammals can acquire **natural** passive immunity to infectious diseases, such as influenza.

- 1
 - 2

[2]

[Total: 14]

224. 9700_s18_qp_22 Q: 6

Enzyme inhibitors and monoclonal antibodies can be used in the treatment of disease.

- (a)** Mevinolin is an enzyme inhibitor that can be prescribed as a drug to reduce the concentration of cholesterol in blood plasma.

High concentrations of cholesterol in the blood have been linked to an increased risk of cardiovascular disease.

Mevinolin acts as a competitive inhibitor of the enzyme HMG CoA reductase. This enzyme catalyses one of the first steps in the synthesis of cholesterol, as shown in Fig. 6.1.

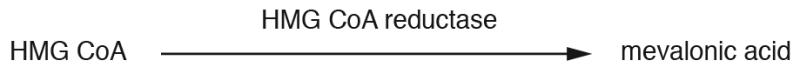


Fig. 6.1

Explain how mevinolin inhibits the enzyme HMG CoA reductase.

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- (b)** Outline the use of monoclonal antibodies in the treatment of disease.

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[Total: 5]

225. 9700_s18_qp_23 Q: 3

Nerium oleander is a xerophytic plant. A photomicrograph of a section through the leaf of *N. oleander* is shown in Fig. 3.1.

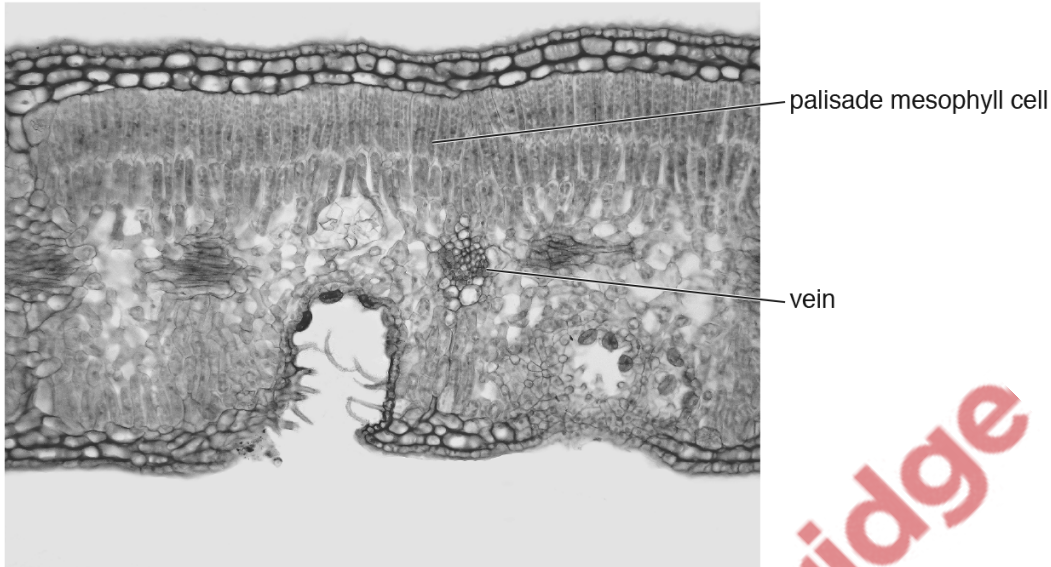


Fig. 3.1

(a) The leaf shown in Fig. 3.1 has a number of adaptations to reduce water loss by transpiration. Two of these adaptations are:

- a multilayered epidermis
- stomata only found in depressions, known as stomatal crypts, on the lower surface of the leaf.

Explain how a multilayered epidermis and stomatal crypts will help to reduce water loss in *N. oleander*.

multilayered epidermis

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stomatal crypts

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.....[3]

Sucrose, amino acids and other assimilates synthesised in palisade mesophyll cells of *N. oleander* pass to the vein, where they can be transported within specialised cells from the source to the sink.

(b) Name the cells specialised for the transport of assimilates in *N. oleander*.

.....[1]

(c) Explain the difference between a source and a sink.

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.....[2]

PapaCambridge

One of the enzymes involved in the synthesis of sucrose in the cytoplasm of palisade mesophyll cells is known as cyFBPase. The gene coding for this enzyme is *cyFBP*.

The importance of cyFBPase in plant growth can be investigated using plants with a mutation in gene *cyFBP*. These plants cannot synthesise cyFBPase.

(d) (i) State what is meant by a gene mutation.

.....
.....[1]

(ii) Suggest **one** way in which the mutation of *cyFBP* prevents the synthesis of cyFBPase.

.....
.....[1]

(e) Monoclonal antibody can be produced that is specific to cyFBPase. This antibody is used by investigators to check that the plants with the *cyFBP* mutation do not synthesise this enzyme.

(i) In monoclonal antibody production, a small mammal is inoculated with cyFBPase and several weeks later cells are removed from the spleen. Some of these cells are required for the production process.

Describe the events occurring within the body of the small mammal that lead to the formation of the cells needed for monoclonal antibody production.

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.....[4]

- (ii) Anti-cyFBPase monoclonal antibody is added to extracts taken from the leaves of the plants with the *cyFBP* mutation.

State the expected results following addition of the monoclonal antibody that would confirm the **absence** of cyFBPase in the leaf extracts.

.....
.....[1]

- (f) Investigations have shown that plants with the *cyFBP* mutation grow to a much smaller height and have proportionately far less starch stored in their roots than normal plants.

Suggest why plants with the *cyFBP* mutation will store less starch in their roots.

.....
.....
.....
.....
.....[2]

[Total: 15]

PapaCambridge

226. 9700_w18_qp_21 Q: 1

Fig. 1.1 shows the human gas exchange system.

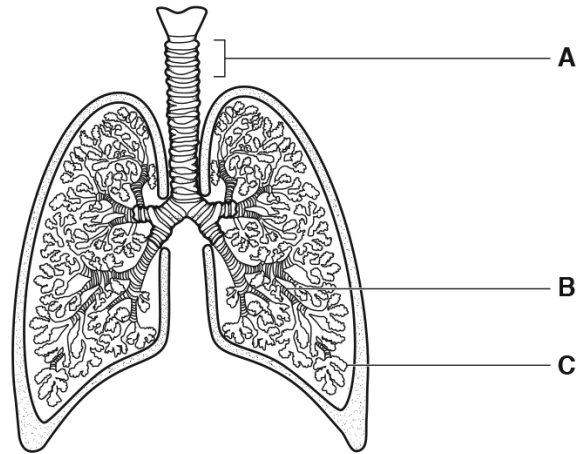


Fig. 1.1

(a) Name the structures labelled A, B, and C in Fig. 1.1.

- A
- B
- C [3]

(b) Name a **non-infectious** disease that affects the human gas exchange system.

..... [1]

(c) Malaria is an infectious disease.

Name the pathogen that causes malaria.

..... [1]

(d) There are a number of vaccines being developed to help control the spread of malaria.

Explain why vaccination programmes have **not** been able to eradicate malaria.

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.....
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..... [3]

(e) Fig. 1.2 shows the distribution of malaria in the Americas in 2012.



Fig. 1.2

Suggest the factors, **other than** lack of vaccines, that could be restricting the distribution of malaria to area P.

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.....
..... [4]

[Total: 12]

227. 9700_w18_qp_21 Q: 4

Fig. 4.1 is a photomicrograph of a cross-section of a tubular structure in the kidney made from epithelial cells.

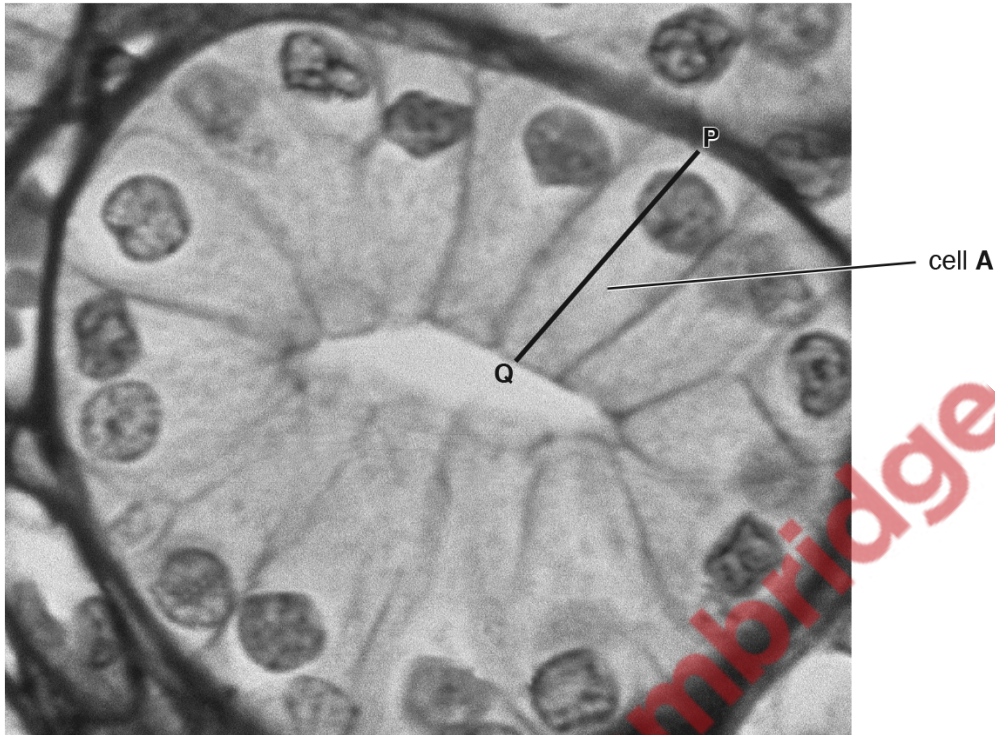


Fig. 4.1

- (a) The actual length of epithelial cell **A** along the line **P–Q** is $35\ \mu\text{m}$.

Calculate the magnification of the image shown in Fig. 4.1. Write down the formula and use it to make your calculation. Show your working.

formula

magnification \times [2]

- (b) Some epithelial cells in the kidney release the protein vascular endothelial growth factor (VEGF). This protein is a cell signalling molecule that stimulates cell division in endothelial cells in blood vessels.

(i) State what occurs during interphase to prepare a cell for division.

.....
.....
.....
.....
..... [2]

(ii) Explain how a cell signalling molecule, such as VEGF, can lead to a response in a cell.

.....
.....
.....
..... [2]

- (c) Uncontrolled cell division may result in a tumour. Tumour cells in the kidney respond to VEGF.

Kidney cancer can be treated with monoclonal antibodies. These monoclonal antibodies bind to VEGF.

Outline the hybridoma method for the production of monoclonal antibodies that will target the VEGF protein.

.....
.....
.....
.....
..... [4]

- (d) Monoclonal antibodies used as a treatment need to be given more than once. Repeated treatment can cause side effects to the person or can become less effective.

Suggest why repeated treatment with monoclonal antibodies may have these effects.

.....
.....
..... [1]

[Total: 11]

228. 9700_w18_qp_22 Q: 4

Viruses share common structural features. Some viruses, such as human immunodeficiency virus (HIV), also have an outer envelope as part of their structure.

(a) Outline the key structural features of viruses.

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.....
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.....
.....
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..... [3]

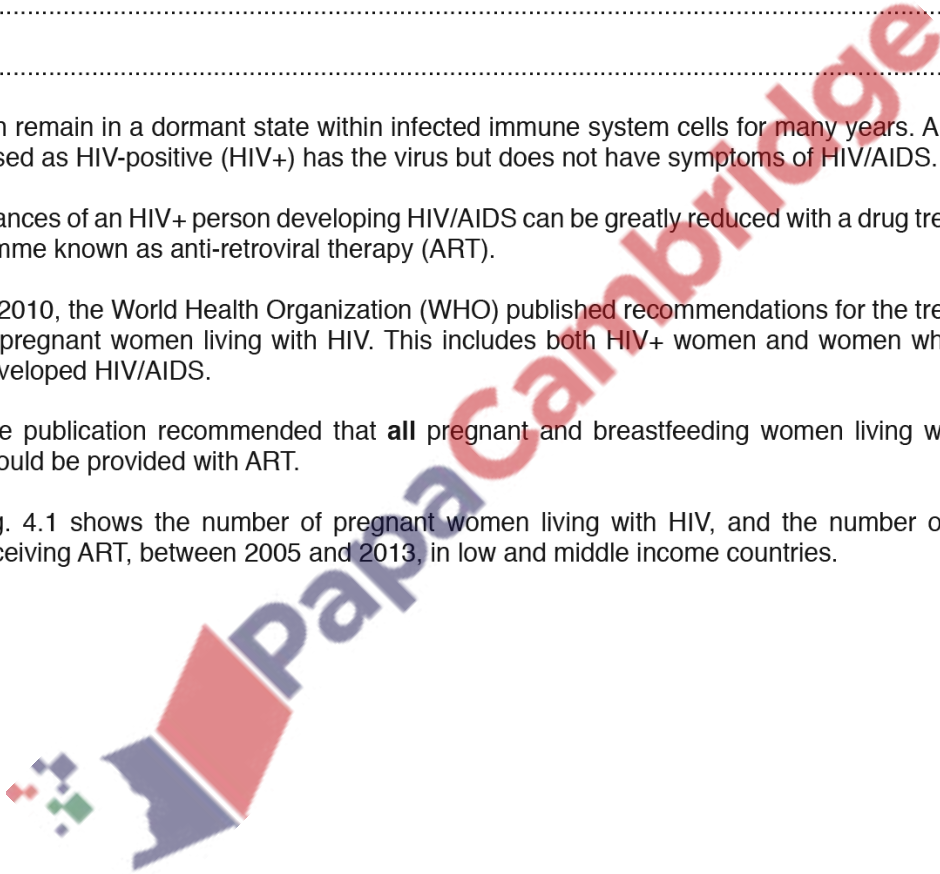
HIV can remain in a dormant state within infected immune system cells for many years. A person diagnosed as HIV-positive (HIV+) has the virus but does not have symptoms of HIV/AIDS.

The chances of an HIV+ person developing HIV/AIDS can be greatly reduced with a drug treatment programme known as anti-retroviral therapy (ART).

(b) In 2010, the World Health Organization (WHO) published recommendations for the treatment of pregnant women living with HIV. This includes both HIV+ women and women who have developed HIV/AIDS.

The publication recommended that **all** pregnant and breastfeeding women living with HIV should be provided with ART.

Fig. 4.1 shows the number of pregnant women living with HIV, and the number of these receiving ART, between 2005 and 2013, in low and middle income countries.



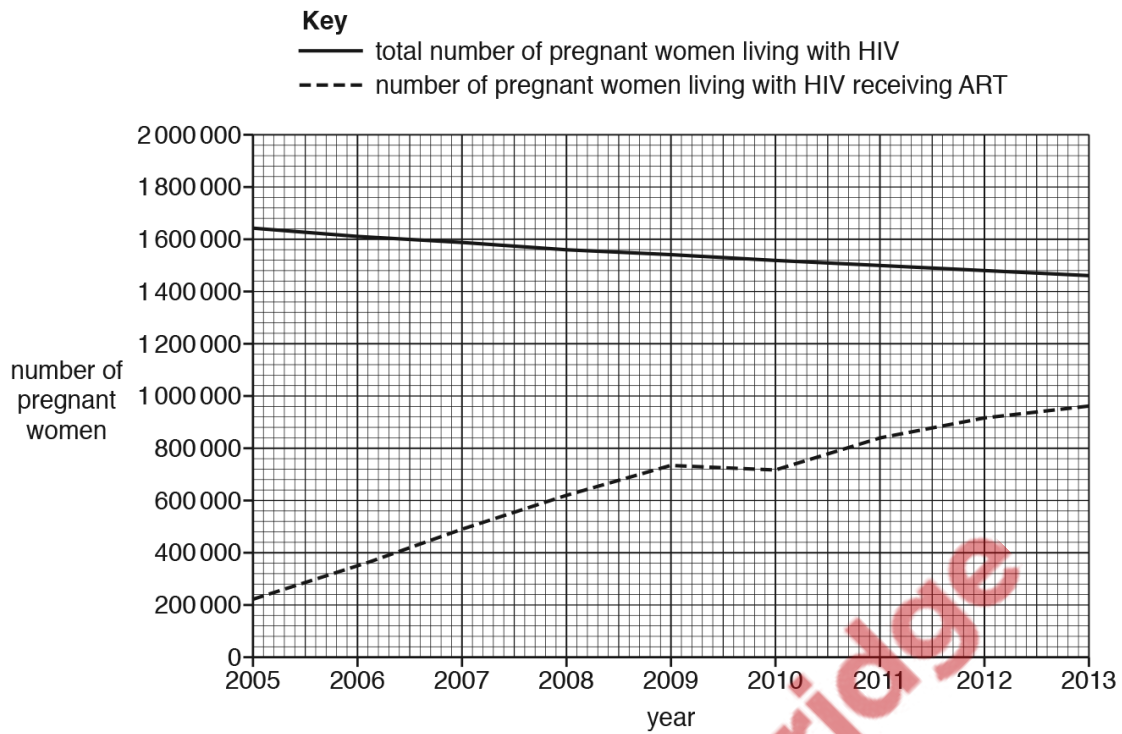


Fig. 4.1

- (i) From the data in Fig. 4.1, it can be calculated that 13% of pregnant women living with HIV received ART in 2005.

Calculate the percentage of pregnant women living with HIV that received ART in 2013.

answer = % [1]

- (ii) Describe the trends shown in Fig. 4.1.

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.....

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..... [3]

- (c) In a person who has been infected with HIV-1, the most common strain of HIV, a sample of blood can be tested for the presence of the virus. One test that can only be used in the early stages of infection involves a monoclonal antibody specific for p24, a structural protein present in the virus.

Fig. 4.2 is a flow chart outlining the steps in the production of anti-HIV p24 monoclonal antibody.

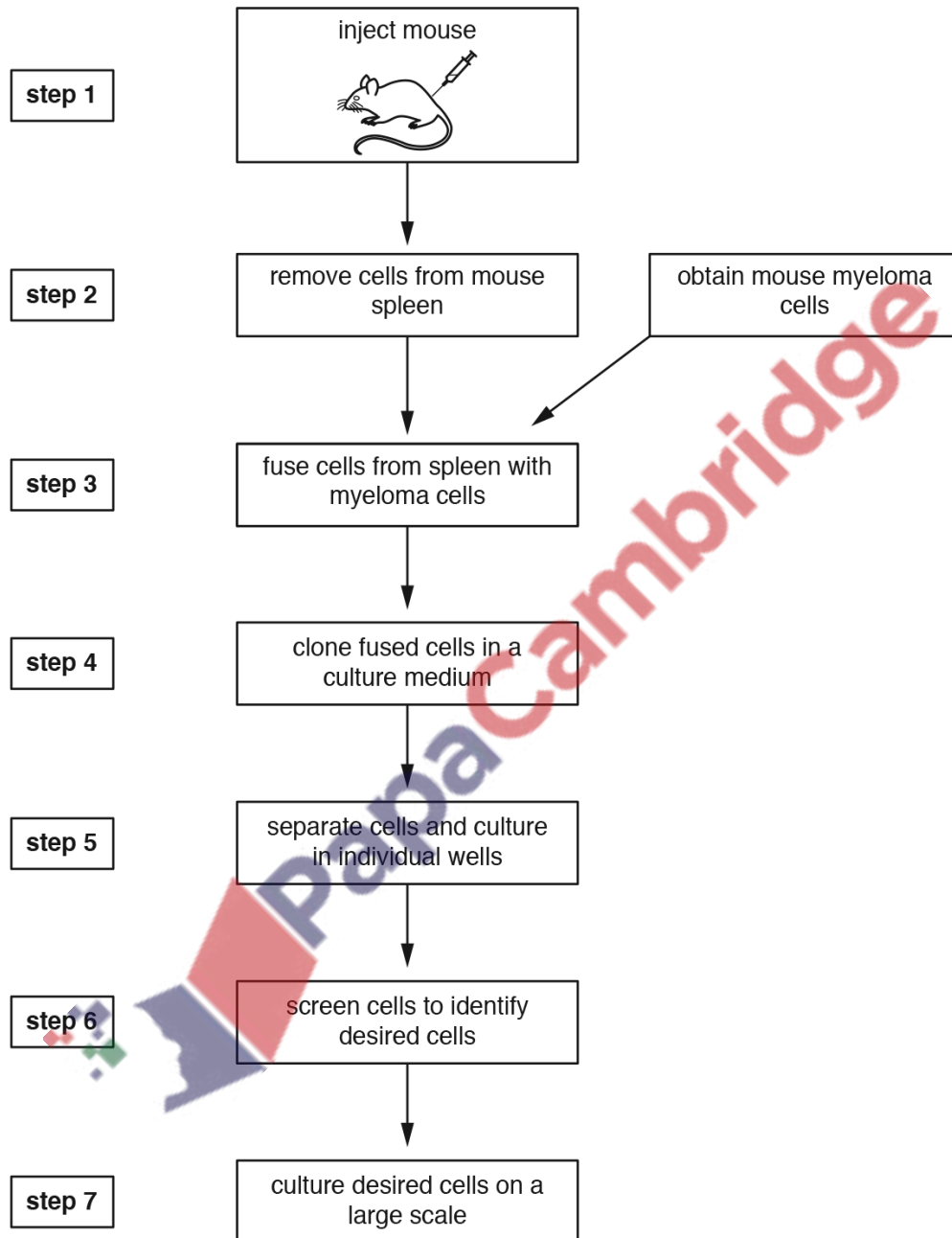


Fig. 4.2

(i) State what is being injected into the mouse in **step 1**.

..... [1]

(ii) Explain why several weeks, rather than several days, separates **step 1** and **step 2**.

.....
.....
..... [1]

(iii) State **one** feature of the myeloma mouse cells, used in **step 3**, that is essential for this production process.

.....
.....
..... [1]

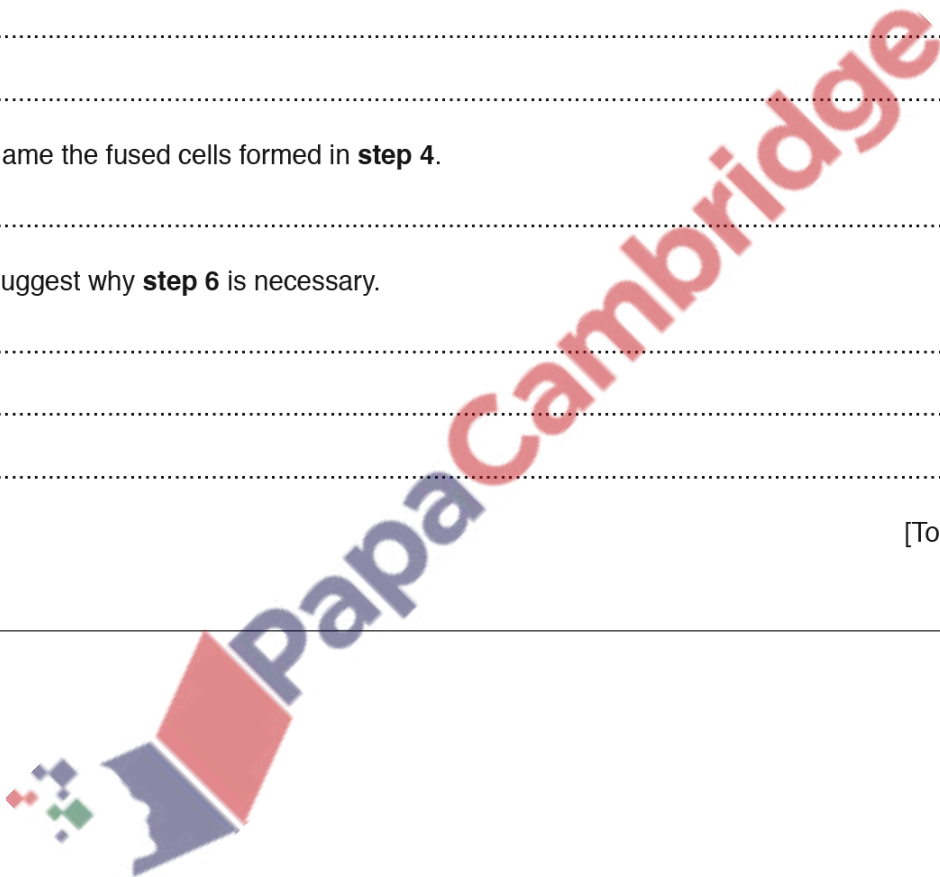
(iv) Name the fused cells formed in **step 4**.

..... [1]

(v) Suggest why **step 6** is necessary.

.....
.....
..... [1]

[Total: 15]



229. 9700_w18_qp_23 Q: 6

- (a) As part of a study of the mitotic cell cycle, a student made stained sections of a root tip of onion, *Allium cepa*, and observed them with a light microscope.

The student made drawings of six of the cells, **A** to **F**, using the high power of the microscope, as shown in Fig. 6.1.

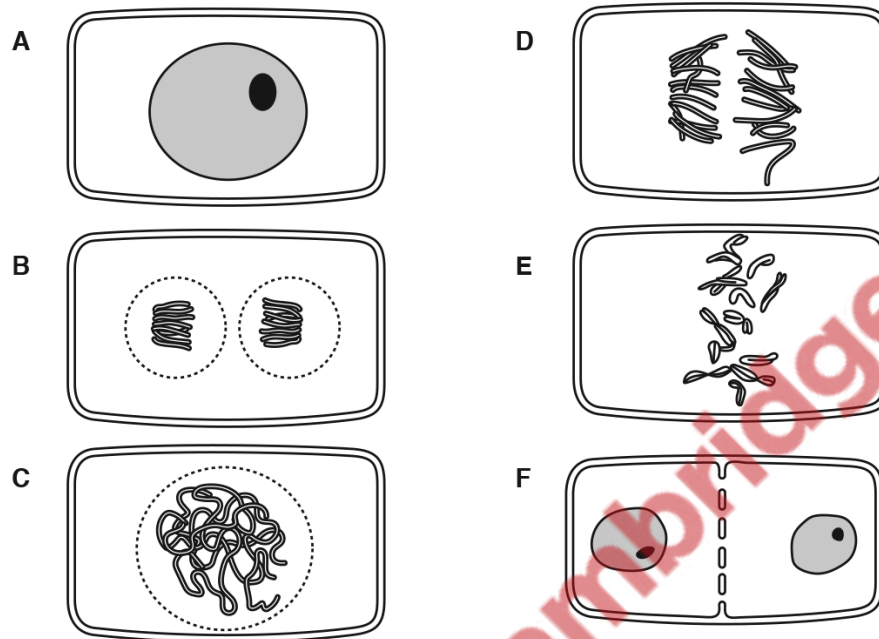


Fig. 6.1

- (i) Complete Table 6.1 to show the sequence of stages in the mitotic cell cycle, using the letters, **A** to **F**, as shown in Fig. 6.1.

Table 6.1

sequence of stages	cell
1	A
2	
3	
4	
5	
6	

[1]

(ii) Table 6.2 shows some events that occur during the mitotic cell cycle in *A. cepa*.

Complete Table 6.2 by naming the stage of the cell cycle when each event occurs.

Table 6.2

event in the cell cycle	name of the stage in the cell cycle
DNA replication	
division of centromeres	
condensation of chromatin	
contraction of spindle fibres	
organisation of chromosomes at the equator	metaphase

[4]

(b) Explain the importance of mitosis in the immune response.

.....

.....

.....

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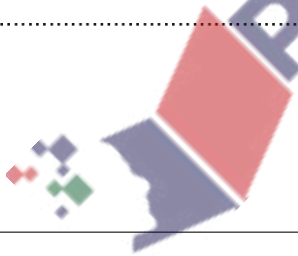
.....

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.....

..... [3]

[Total: 8]



230. 9700_m17_qp_22 Q: 2

The infectious disease cholera is caused by a bacterium.

(a) Fig. 2.1 shows a transmission electron micrograph of this bacterium.

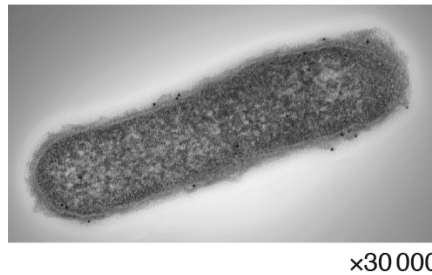


Fig. 2.1

(i) Name the bacterium that causes cholera.

..... [1]

(ii) The bacterium in Fig. 2.1 is an example of a prokaryotic cell.

Each of the descriptions **A** to **C** describes a cell structure found in prokaryotic cells **and** in plant cells.

For each of the descriptions **A** to **C**:

- name the cell structure described
- state **one** difference in this structure between a prokaryotic cell and a plant cell.

A the site of polypeptide synthesis

cell structure

difference

.....

B the genetic material of the cell

cell structure

difference

.....

C the structure that provides a rigid shape to the cell and prevents osmotic lysis

cell structure

difference

.....

[6]

(b) Cholera is an example of an infectious disease.

Explain what is meant by an infectious disease.

.....
.....
.....
..... [2]

The symptoms of cholera are caused by cholera toxin, a toxin released by the bacterium.

Cholera toxin is a protein made up of six polypeptides:

- a single copy of a polypeptide known as the A subunit that includes an extended alpha helix
- five polypeptides that together make the B subunit.

The B subunit of cholera toxin binds to a cell surface membrane component, known as GM1, of an intestinal epithelial cell. The complete cholera toxin protein then enters the cell by endocytosis. Once inside the cell, the A subunit of the protein acts as an enzyme, disrupting the normal functioning of the cell.

(c) List the levels of protein structure present in cholera toxin.

.....
..... [2]

(d) Outline the mechanism by which cholera toxin enters the cell.

You may use the space for annotated diagrams.

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..... [3]

- (e) Using genetic engineering, it is possible to produce a form of cholera toxin consisting of only subunit B. This can be combined with inactivated bacterial cells to produce a vaccine against cholera.

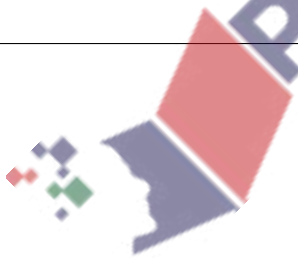
- (i) Suggest why subunit B, rather than subunit A, is used in the vaccine.

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..... [1]

- (ii) Outline how this vaccine can give protection against cholera.

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..... [5]

[Total: 20]



231. 9700_s17_qp_21 Q: 3

Fig. 3.1 is a diagram that shows the structure of an antibody molecule.

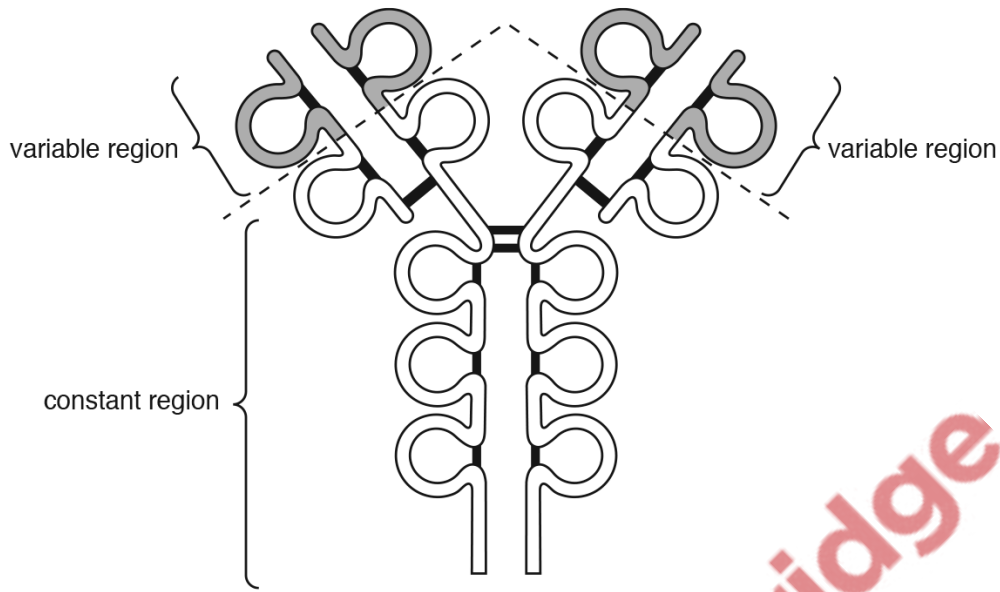


Fig. 3.1

(a) State why the antibody molecule shown in Fig. 3.1 has quaternary structure.

.....
.....[1]

(b) (i) Use Fig. 3.1 to explain how the structure of the variable region of an antibody molecule is related to its function.

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.....[3]

(ii) State the role of the constant region of an antibody.

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.....[1]

(c) Monoclonal antibodies are used both in diagnosis and in treatment of disease.

(i) Outline how monoclonal antibodies are produced.

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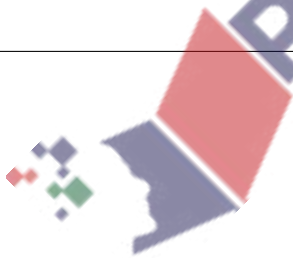
[4]

(ii) Suggest the advantages of using monoclonal antibodies in diagnosis of disease.

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[2]

[Total: 11]



232. 9700_w17_qp_21 Q: 3

Cells of the immune system function to protect the body against infectious diseases.

(a) (i) Name the type of cell that produces antibodies.

.....[1]

(ii) The virus that causes the infectious disease influenza has two antigens, **H** and **N**. Antibodies are produced in response to an infection by this virus. The antibodies are specific for either antigen **H** or for antigen **N**.

Describe how the structure of an antibody molecule allows it to be specific for **one** antigen, such as **H** or **N**.

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.....[3]

(b) Cholera is a disease caused by a bacterial pathogen.

(i) Name the pathogen that causes cholera.

.....[1]

(ii) Describe how the pathogen that causes cholera is transmitted.

.....
.....
.....
.....[2]

- (c) Viruses that infect bacteria are called bacteriophages. Some bacteriophages that infect the cholera pathogen cause lysis of the bacterium.

- (i) Suggest what happens to the structure of a bacterial cell to cause lysis.

.....
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.....
.....
.....[2]

- (ii) Some scientists believe that bacteriophages could be used to treat people who are infected with cholera.

Suggest the properties of the bacteriophages that would make this possible.

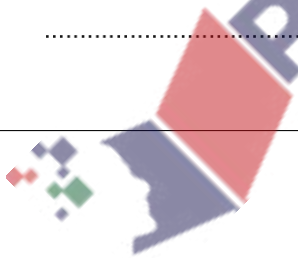
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.....[2]

- (iii) Antibiotics can be used to treat people with cholera.

State why antibiotics are **not** effective against measles.

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.....[1]

[Total: 12]



233. 9700_w17_qp_23 Q: 4

Malaria is a disease transmitted by a vector.

(a) (i) State the name of the pathogen that causes malaria.

.....[1]

(ii) State the name of the vector that transmits the pathogen.

.....[1]

In 2014, the World Health Organization (WHO) estimated that 3200 million people were at risk of malaria. This was almost half of the world population in 2014.

Table 4.1 shows the number of cases of malaria and the number of deaths from malaria between 1998 and 2013. The table shows numbers for all the countries of the world and for the countries in the WHO African region.

The table also shows the numbers in the African region as percentages of the numbers for all countries.

Table 4.1

year	number of cases of malaria in millions		cases in the African region as a percentage of all countries	number of deaths from malaria in thousands		deaths in the African region as a percentage of all countries
	all countries	African region		all countries	African region	
1998	272.9	237.6	87.1	1110.0	961.0	90.1
2003	236.0	186.6	79.1	872.0	800.0	91.7
2008	225.1	181.0	80.4	747.0	677.0	90.6
2013	198.0	158.4	80.0	584.0	525.6	90.0

(b) Describe the trends shown in Table 4.1.

.....

[3]

- (c) Suggest reasons why the number of cases of malaria and the number of deaths from malaria changed between 1998 and 2013.

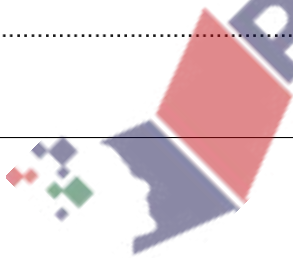
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.....[3]

- (d) Malaria is very difficult to control even though there is improved understanding of the disease.

Explain why malaria is very difficult to control.

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.....[4]

[Total: 12]



(c) The structure of *Morbillivirus* is shown in Fig. 4.1.

Haemagglutinin (**H**) and fusion protein (**F**) are glycoproteins embedded in the viral envelope.

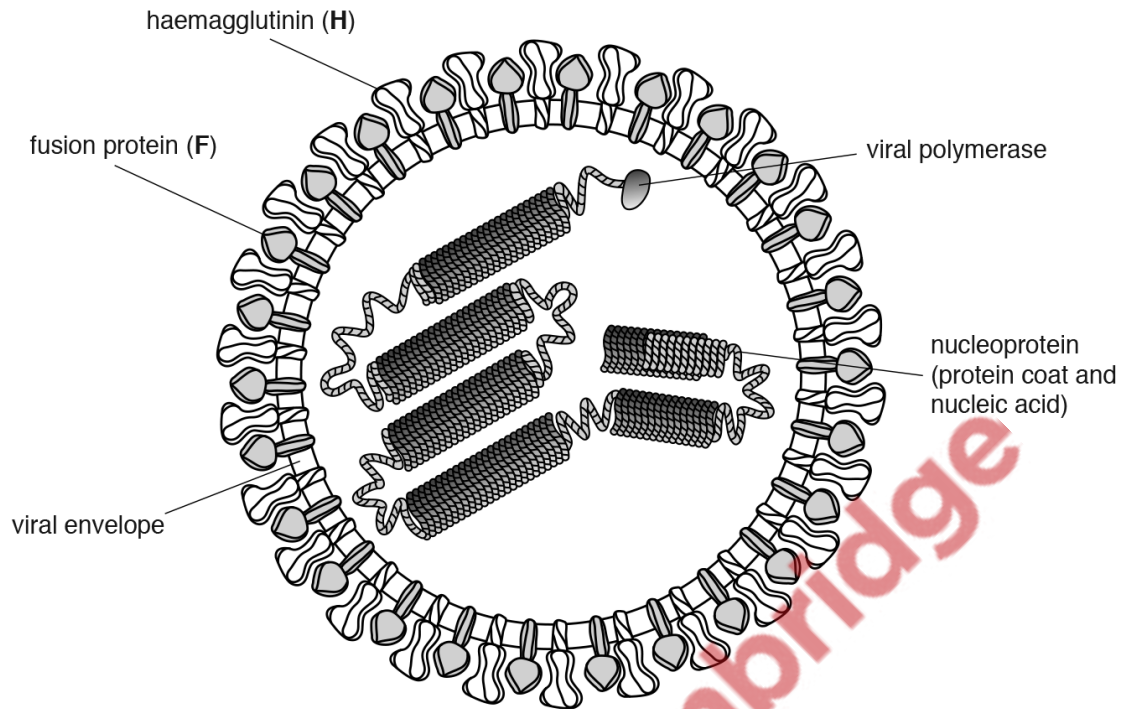


Fig. 4.1

Morbillivirus only infects cells that have a membrane glycoprotein known as signalling lymphocyte activation molecule (SLAM).

When *Morbillivirus* infects a cell, **H** acts before **F**. After the virus binds to the host cell, only the nucleoprotein with the viral polymerase enters the host cell and the virus is replicated.

New viral particles leave the host cell by budding from the cell surface membrane of the cell. This forms the main part of their envelope.

With reference to Fig. 4.1 and the information provided on pages 9 and 10,

- (i) outline the structural features of the viral envelope of *Morbillivirus*

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.....[2]

- (ii) suggest how *Morbillivirus* infects a cell with SLAM glycoproteins so that only nucleoprotein and viral polymerase enter

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.....
.....[3]

- (iii) suggest the role of viral polymerase in *Morbillivirus*.

.....
.....
.....
.....[2]

(d) HIV has an antigen known as p24.

One test for an early diagnosis of HIV infection uses a monoclonal antibody that identifies antigen p24.

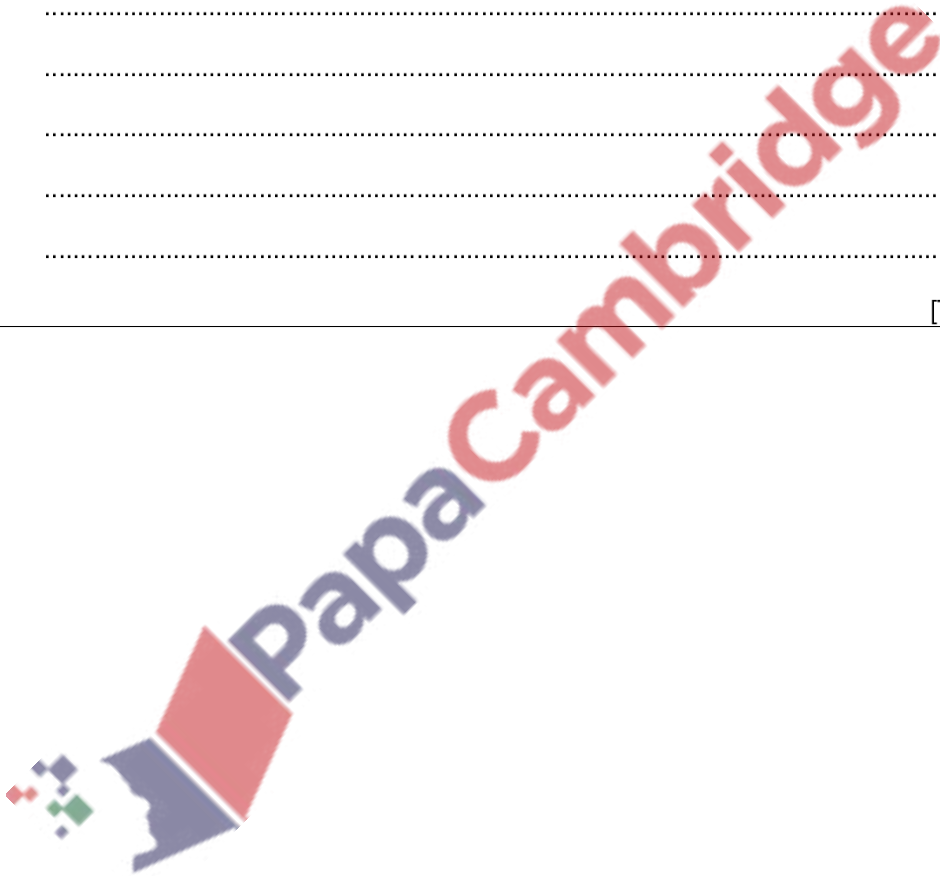
(i) State the type of biological molecule that is represented by antigen p24.

.....[1]

(ii) Outline how the monoclonal antibody against antigen p24 is produced.

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.....
.....[3]

[Total: 17]



235. 9700_s16_qp_23 Q: 3

Rheumatoid arthritis (RA) is a disease of the joints in the human body.

- (a) RA is classed as an auto-immune disease where the immune system treats some self antigens as non-self.

Explain what is meant by the term *non-self antigens*.

.....
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.....
.....
.....
.....
.....[3]

- (b) The symptoms of RA include inflammation of the joints which causes pain and difficulty in movement of the joint.

The inflammation is triggered by a chemical known as $TNF-\alpha$, produced by macrophages.

One approach to the treatment of RA is by the use of monoclonal antibody against $TNF-\alpha$.

Fig. 3.1 is a diagram of an antibody molecule.

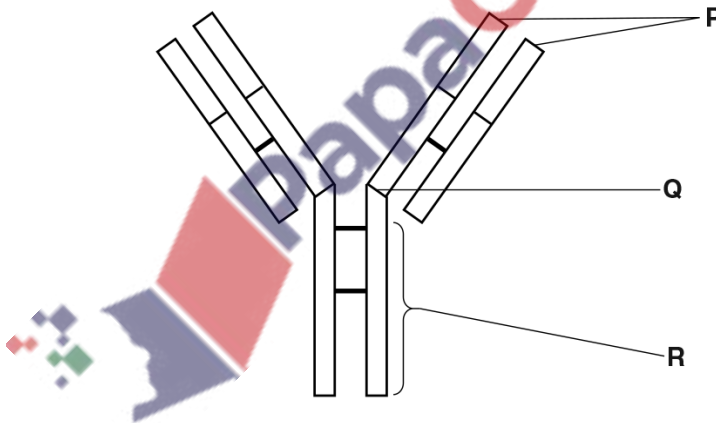


Fig. 3.1

- (i) Name the parts of the antibody molecule labelled P, Q and R.

P

Q

R

[3]

- (ii) Name the type of bonds that hold the polypeptide chains together in the antibody structure.

.....[1]

- (c) (i) Outline how monoclonal antibody against TNF- α is produced.

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.....
.....
.....[3]

- (ii) Suggest how monoclonal antibody against TNF- α can reduce the symptoms of RA.

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.....
.....
.....[2]

[Total: 12]



236. 9700_w16_qp_21 Q: 5

- (a) Monoclonal antibodies (MAbs) have been used in the treatment of some non-infectious diseases.

MAbs can be designed to bind to a protein on diseased cells, so causing their destruction by cells of the person's immune system.

- (i) Name the part of an antibody molecule that will bind to a protein on diseased cells.

.....[1]

- (ii) Suggest how the binding of monoclonal antibody to the diseased cells causes their destruction by cells of the person's immune system.

.....
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.....
.....
.....
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.....
.....
.....
.....
.....
.....
.....[4]

- (b) Myasthenia gravis is an auto-immune disease.

Explain the term *auto-immune disease*.

.....
.....
.....
.....
.....
.....[1]

[Total: 6]

(b) Fig. 5.1 is a summary of some infectious diseases.

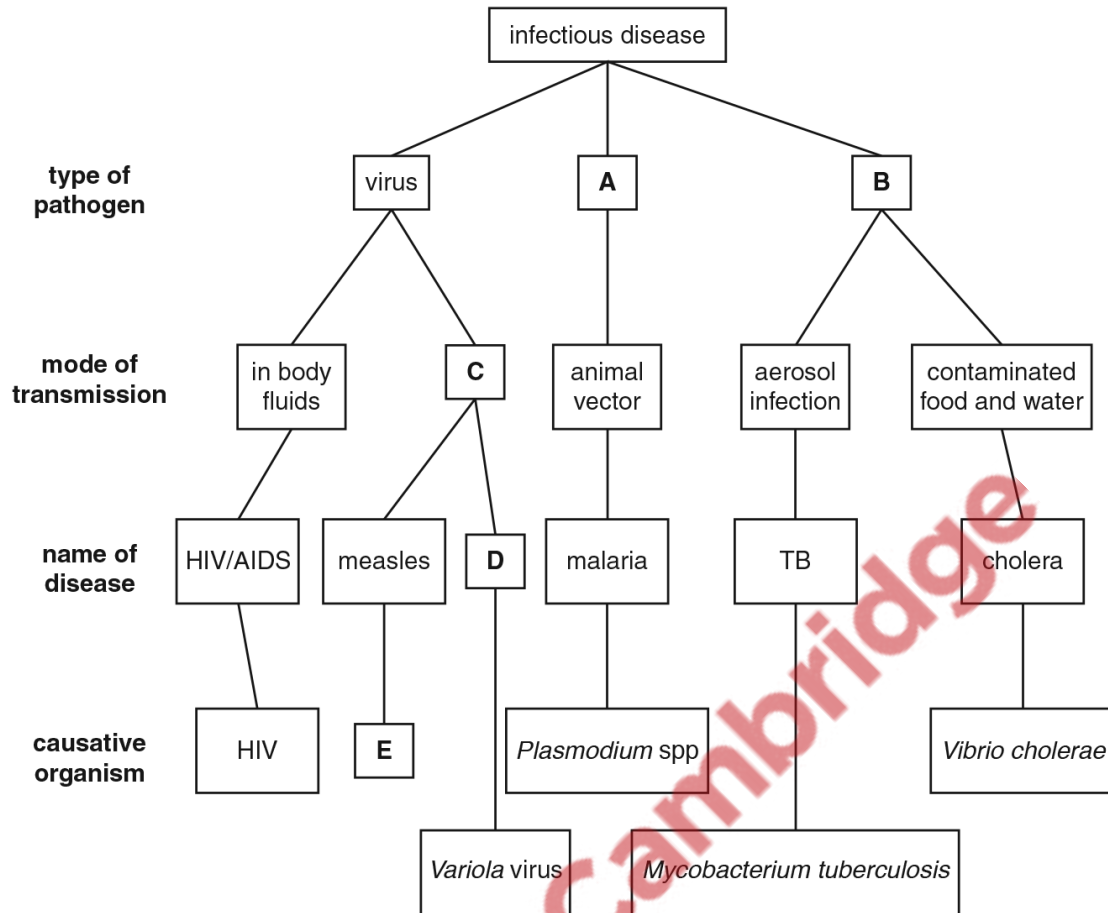


Fig. 5.1

Use the information in Fig. 5.1 to answer parts (i) to (iv).

(i) Name the type of pathogen represented by **A** and **B**.

A

B

[2]

(ii) State the mode of transmission represented by **C**.

.....[1]

(iii) Name the disease represented by **D**.

.....[1]

(iv) Name the causative organism represented by **E**.

.....[1]

238. 9700_w16_qp_23 Q: 6

Measles is a highly infectious disease.

(a) Name the pathogen that causes measles.

.....[1]

The number of cases of measles is reported to the World Health Organization (WHO) by countries throughout the world so that global data are collected.

Fig. 6.1 shows the global data collected between January 2008 and December 2012.

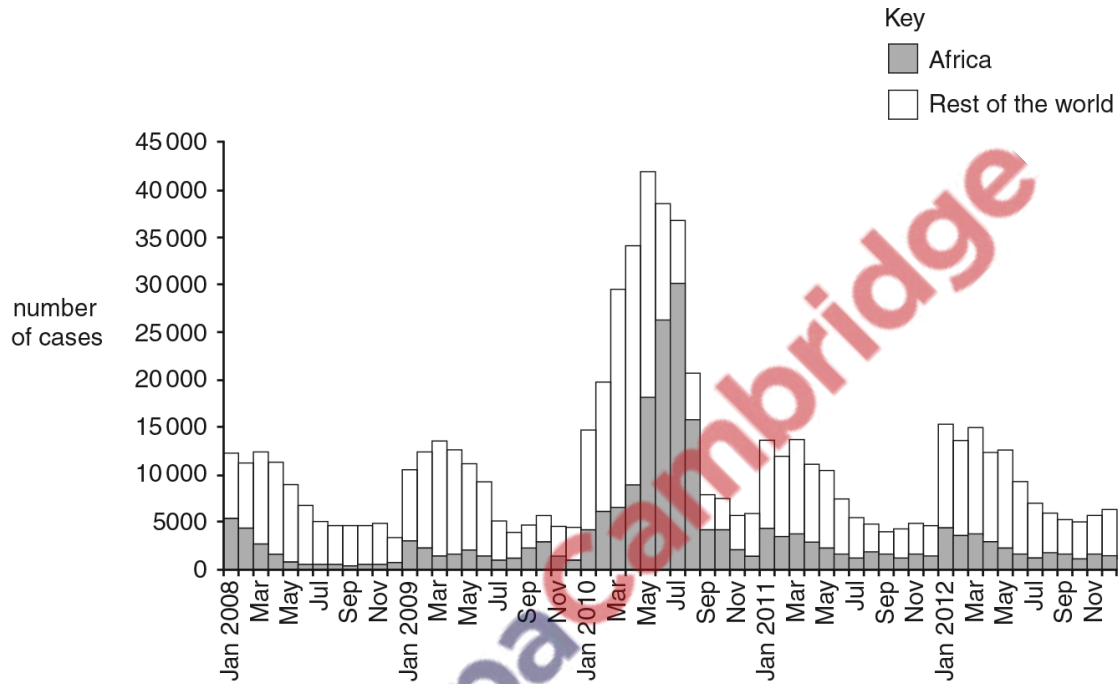


Fig. 6.1

(b) Use the data in Fig. 6.1 to describe the pattern shown in the number of cases of measles reported to the WHO between January 2008 and December 2012.

.....

[3]

239. 9700_s15_qp_22 Q: 5

- (a) Natural immunity and artificial immunity can both be acquired in a passive or in an active manner.

Table 5.1 shows information about immunity acquired by two individuals, **P** and **Q**.

Complete Table 5.1.

Table 5.1

description of event	outcome for the individual	production of memory cells / yes or no	type of immunity acquired by individual
individual P is injected with a live, weakened disease-causing organism	individual P does not become ill from the disease and has long-lasting protection from the disease
individual Q is injected with antibody against a specific disease-causing organism	individual Q does not become ill from the disease but is ill with the disease a year later

[2]

Fig. 5.1 is a light micrograph of a sample of blood. Cell X is a phagocyte.

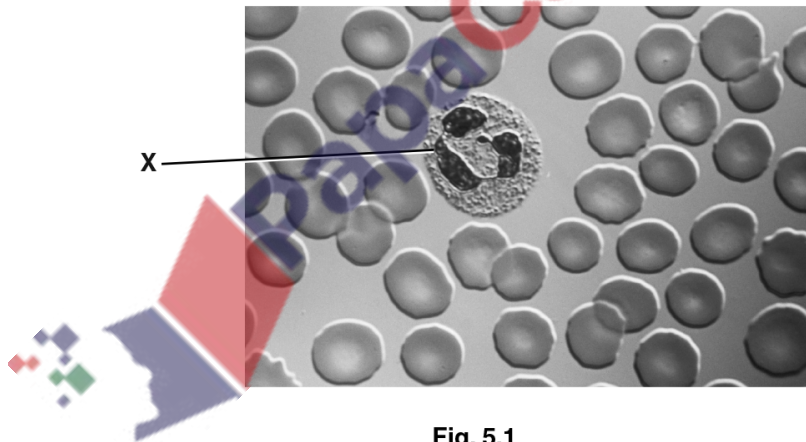


Fig. 5.1

- (b) State the origin of the blood cell labelled X.

.....[1]

- (c) Phagocytes play an important role when an immune response is initiated against cancerous tumour cells.
- (i) Suggest how phagocytes can recognise the difference between healthy body cells and cancerous tumour cells.

.....

.....

.....

.....

.....[2]

- (ii) Outline briefly how a tumour forms.

.....

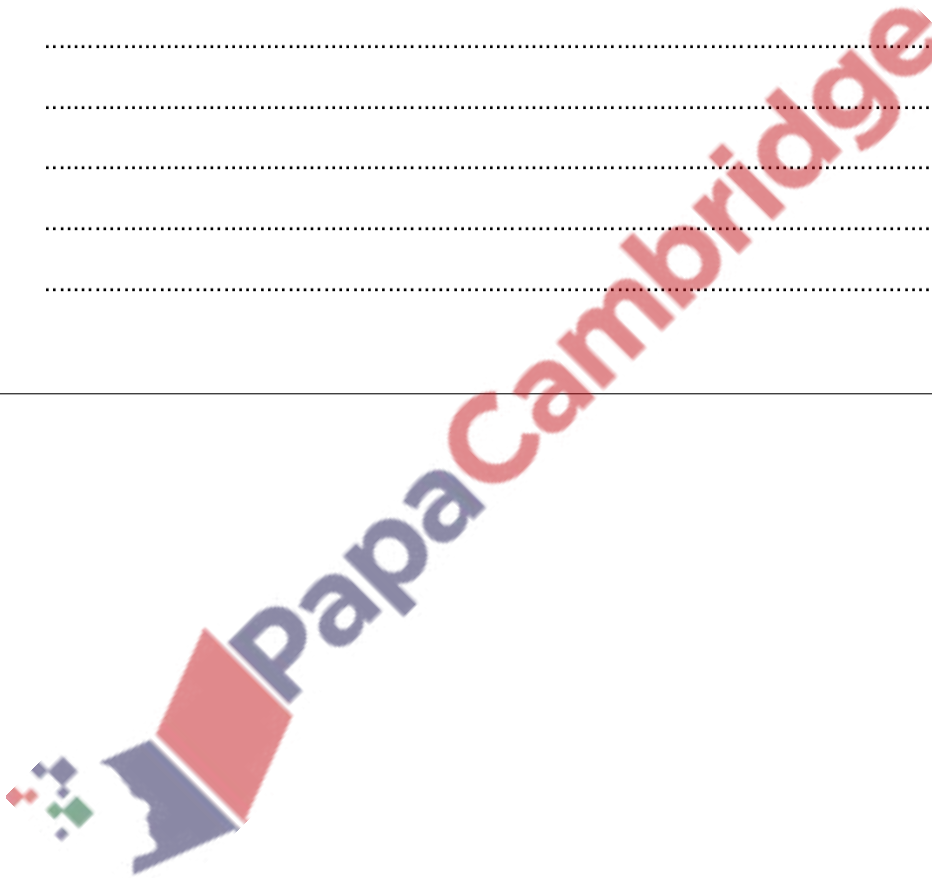
.....

.....

.....

.....[2]

[Total: 7]



240. 9700_s15_qp_23 Q: 3

Fig. 3.1 is an electron micrograph of a type of B-lymphocyte called a plasma cell.

Plasma cells secrete antibody molecules.

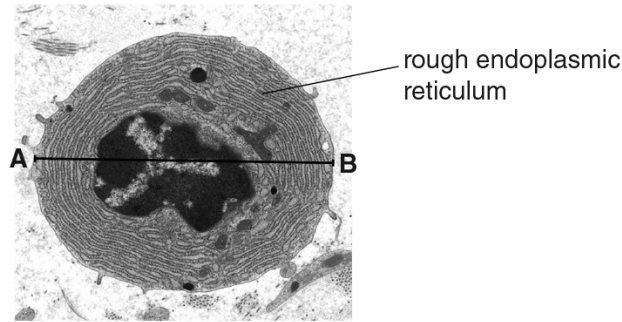


Fig. 3.1

(a) Suggest why plasma cells contain a large quantity of rough endoplasmic reticulum.

.....
.....
.....
.....
.....[2]

(b) The diameter **A – B** of the plasma cell in Fig. 3.1 is $15\mu\text{m}$.

Calculate the magnification of Fig. 3.1.

Show your working.

magnification \times [2]

(c) Smallpox was the first disease to be eradicated by vaccination. The vaccine was effective for up to 10 years after one dose and did not require boosters within this time.

Name the causative organism (pathogen) of smallpox.

.....[1]

- (d) When a person received the smallpox vaccine, the numbers of plasma cells specific for the smallpox pathogen were measured from blood samples taken over a period of 35 days.

Fig. 3.2 shows how the numbers of smallpox-specific plasma cells changed during 35 days after vaccination.

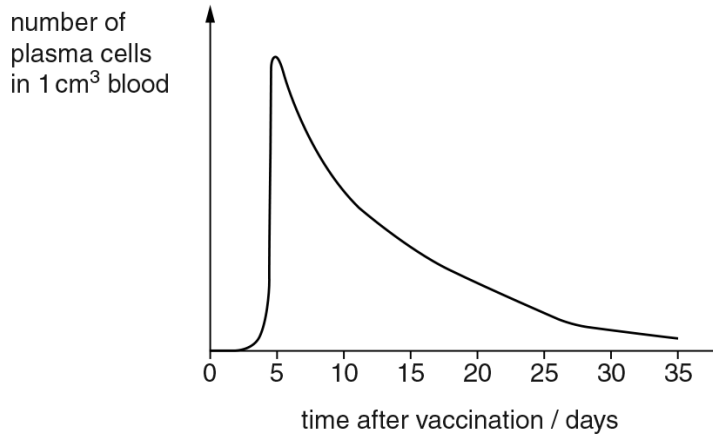


Fig. 3.2

Fig. 3.2 shows that the number of smallpox-specific plasma cells increases and then decreases within 35 days of vaccination.

Explain how a single dose of this vaccine can provide immunity for up to 10 years when the plasma cells are short-lived.

.....

 [3]

- (e) State two reasons why the vaccination programme was successful in eradicating smallpox.

1

 2

 [2]

- (f) State the type of immunity provided by the smallpox vaccine.

..... [1]

[Total: 11]

241. 9700_w15_qp_23 Q: 1

Antibodies are secreted by activated B-lymphocytes known as plasma cells.

Fig. 1.1 is a diagram showing the cellular processes involved in the production of a polypeptide of an antibody molecule (not drawn to scale).

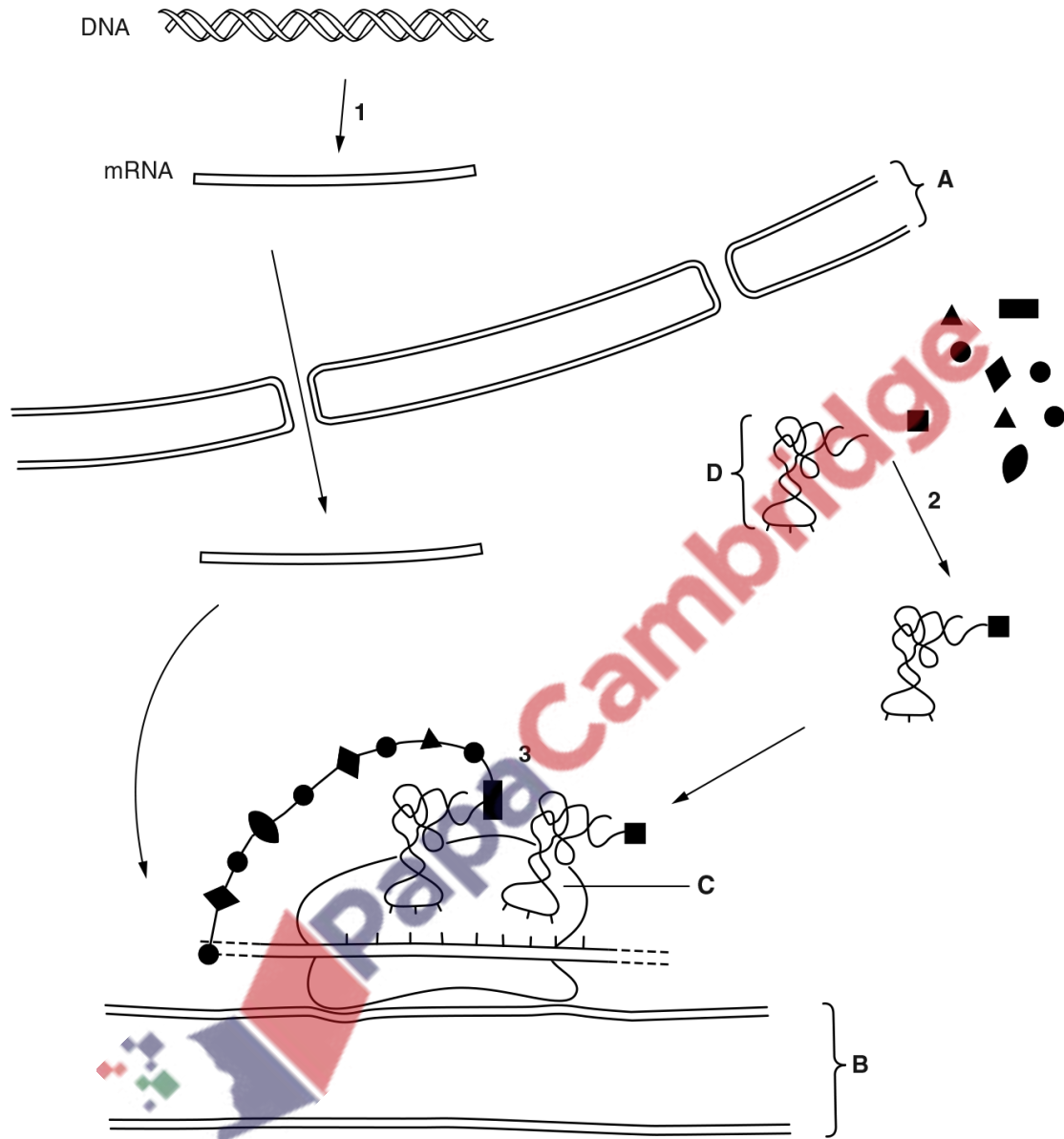


Fig. 1.1

(a) (i) Name structures **A**, **B** and **C**.

A

B

C [3]

(ii) Name molecule **D**.

D [1]

(iii) State what is occurring at **1**, **2** and **3**.

at **1**

.....

at **2**

.....

at **3**

..... [3]

(b) Antibodies are glycoproteins.

State what is meant by the term glycoprotein.

.....

.....

..... [1]

(c) The genes responsible for antibody production are found on different chromosomes, such as chromosomes 2 and 14 in humans.

Explain how one antibody molecule is the product of more than one gene.

.....

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.....

..... [2]

(d) Describe **and** explain how the structure of an antibody molecule is related to its functions.

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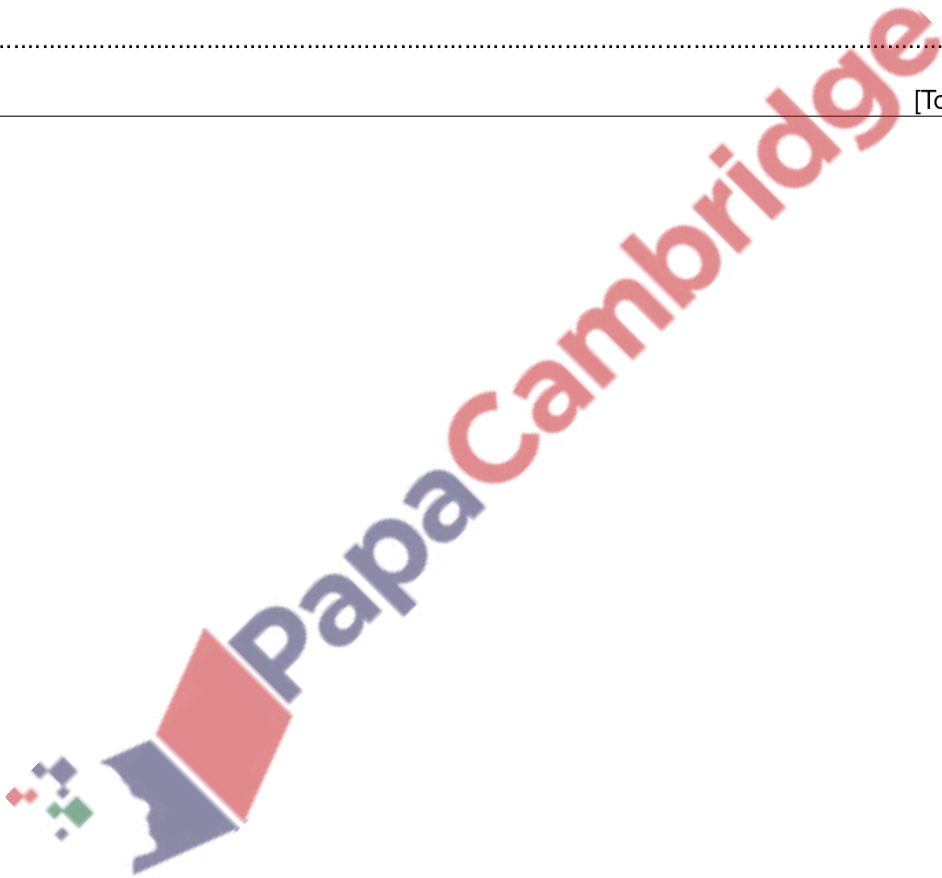
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..... [4]

[Total: 14]



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