Gene Expression and Cancer

These practice questions can be used by students and teachers and is

Suitable for AQA A Level 7402 Biology Topic Question

Level: AQA A LEVEL 7402

Subject: Biology

Exam Board: AQA A Level 7402

Topic: Gene Expression and Cancer

1

(Extra space) _____

(3)

Scientists investigated a possible relationship between the percentage of fat in the diet and the death rate from breast cancer in women from 10 countries.

Their data is shown in the table below.

Percentage of fat in diet of population	Death rate of women from breast cancer per 100 000 women
9.5	1.5
15.0	7.0
20.0	12.0
25.0	9.0
32.0	15.0
35.0	8.0
35.0	20.0
40.5	18.0
43.0	24.0
45.0	26.0

(b) Describe how you would plot a suitable graph of these data. Explain your choice of type ofgraph.

(Extra space) _____

(3)

(c) What can you conclude from these data?

(2)

(Total 8 marks)

2 Metastatic melanoma (MM) is a type of skin cancer. It is caused by a faulty receptor protein in cell-surface membranes. There have been no very effective treatments for this cancer.

Dacarbazine is a drug that has been used to treat MM because it appears to increase survival time for some people with MM.

Doctors investigated the use of a new drug, called ipilimumab, to treat MM. They compared the median survival time (ST) for two groups of patients treated for MM:

- a control group of patients who had been treated with dacarbazine
- a group of patients who had been treated with dacarbazine and ipilimumab.

The ST is how long a patient lives after diagnosis.

The doctors also recorded the percentage of patients showing a significant reduction in tumours with each treatment.

The total number of patients in the investigation was 502.

The table below shows the doctors' results.

Treatment	Median survival time (ST) / months	Percentage of patients showing significant reduction in tumours
Dacarbazine	9.1	10.3
Dacarbazine and ipilimumab	11.2	15.2

(a) The doctors compared median survival times for patients in each group.

How would you find the median survival time for a group of patients?

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(2)

(b) In many trials of new drugs, a control group of patients is given a placebo that does notcontain any drug.

The control group in this investigation had been treated with dacarbazine.
Suggest why they had not been given a placebo.

(1)

(c) A journalist who read this investigation concluded that ipilimumab improved the treatmentof MM.

Do the data in the table support this conclusion? Give reasons for your answer.

(Extra space)

(4)

(d) MM is caused by a faulty receptor protein in cell-surface membranes.
Cells in MM tumours can be destroyed by the immune system.

Suggest why they can be destroyed by the immune system.

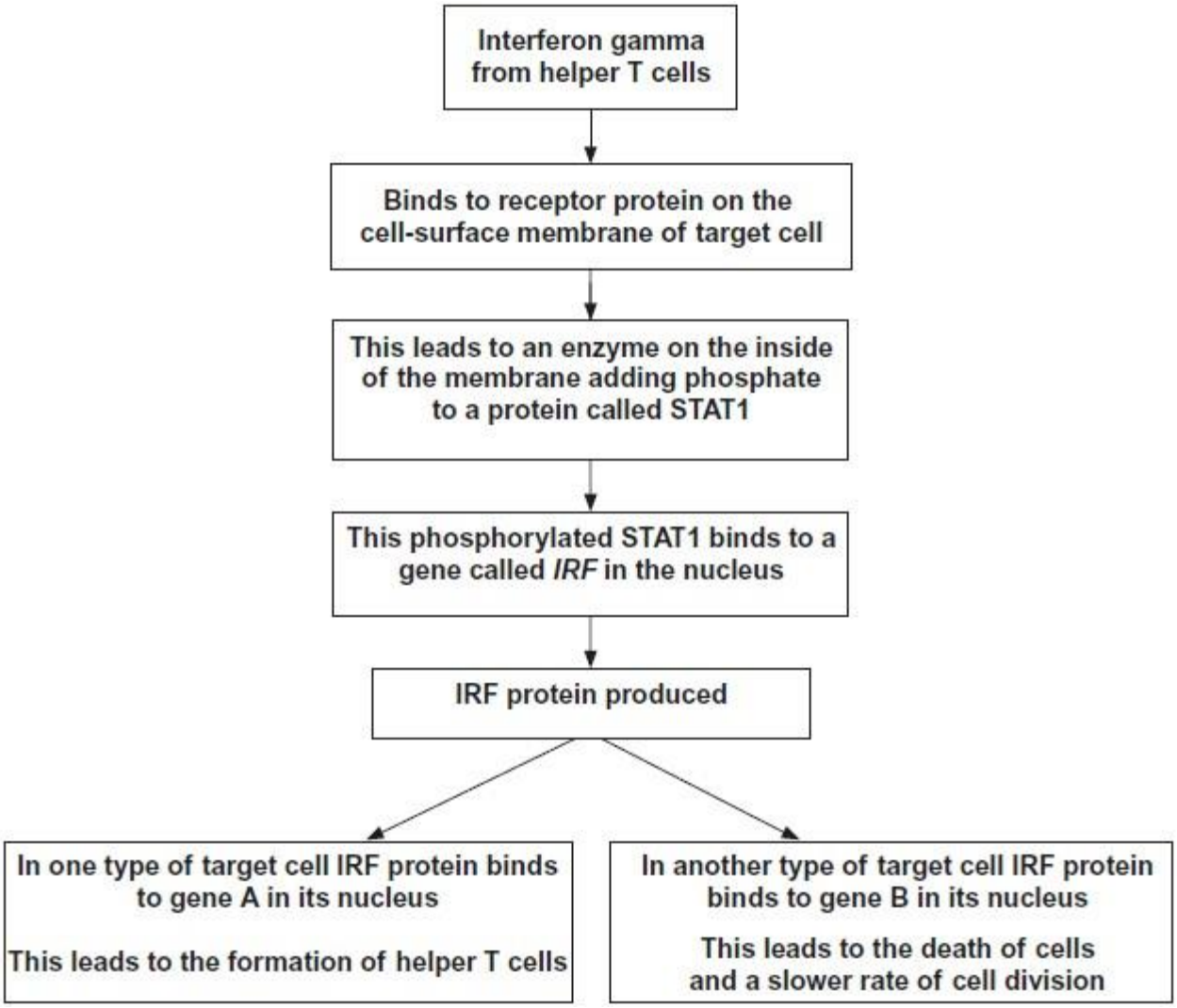
(Extra space) _____

(3)

(Total 10 marks)

3 Interferon gamma is a substance secreted by some types of white blood cells, including helper T cells. It regulates the production of a number of proteins by target cells. Which protein is produced depends on the type of target cell.

The diagram shows how interferon gamma regulates three genes.



(a) Use information in the diagram to suggest how the binding of interferon gamma to itsreceptor protein leads to the production of phosphorylated STAT1.

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(2)

(b) Name the **two** transcription factors in the diagram.

1. _____

2. _____

(2)

(c) The regulation of the formation of helper T cells by interferon gamma is an example ofpositive feedback.

Explain why it is an example of positive feedback.

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(2)

(d) The *IRF* gene can be a tumour suppressor gene.

Use the information in the diagram to explain how the *IRF* gene acts as a tumour suppressor gene.

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(3)

(Total 9 marks)

4 Oestrogen is a substance produced by the enzyme aromatase. In females, the main source of oestrogen is the ovaries but aromatase is produced by many other organs in the body, including the lungs. Oestrogen can stimulate the development of some lung tumours. In these tumours, binding of oestrogen to cell-surface receptors stimulates cell division.

Scientists investigated whether two drugs could prevent lung tumours in female mice. First, they removed the ovaries from these mice. They then injected the mice with a tumour-causing chemical found in tobacco twice a day for 4 weeks. The mice were then randomly allocated to one of four groups. Each group contained 10 mice.

- Group **Q** was given a placebo. This placebo did not contain either drug.
- Group **R** was given the drug anastrozole. This inhibits the enzyme aromatase.
- Group **S** was given the drug fulvestrant. This binds to oestrogen receptors.
- Group **T** was given both anastrozole and fulvestrant.

The mice were given these drugs each week during weeks 5−15 of the investigation.

(a) The scientists removed the ovaries from the mice for the investigation. They also gave themice injections of the substrate of aromatase each day.

Explain why these steps were necessary.

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(2)

(b) The scientists predicted that fulvestrant would be more effective when given withanastrozole than when given alone.

Use the information provided to suggest why they predicted this.

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(2)

At week 15, the lungs of the mice were removed and examined. The scientists then determined the number of tumours present and the mean tumour area for each group.

Figure 1 and **Figure 2** show the scientists' results.

Figure 1

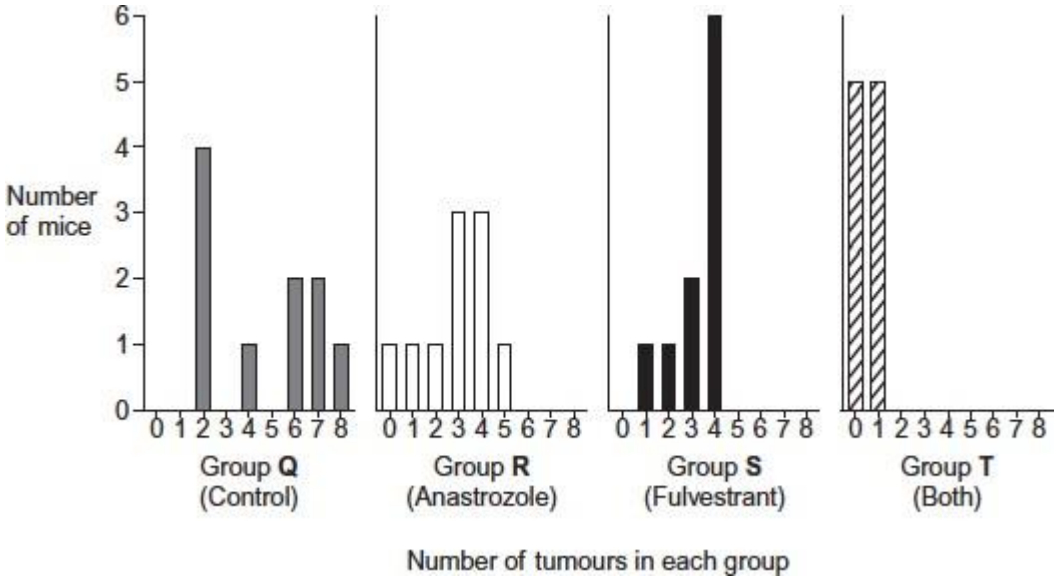
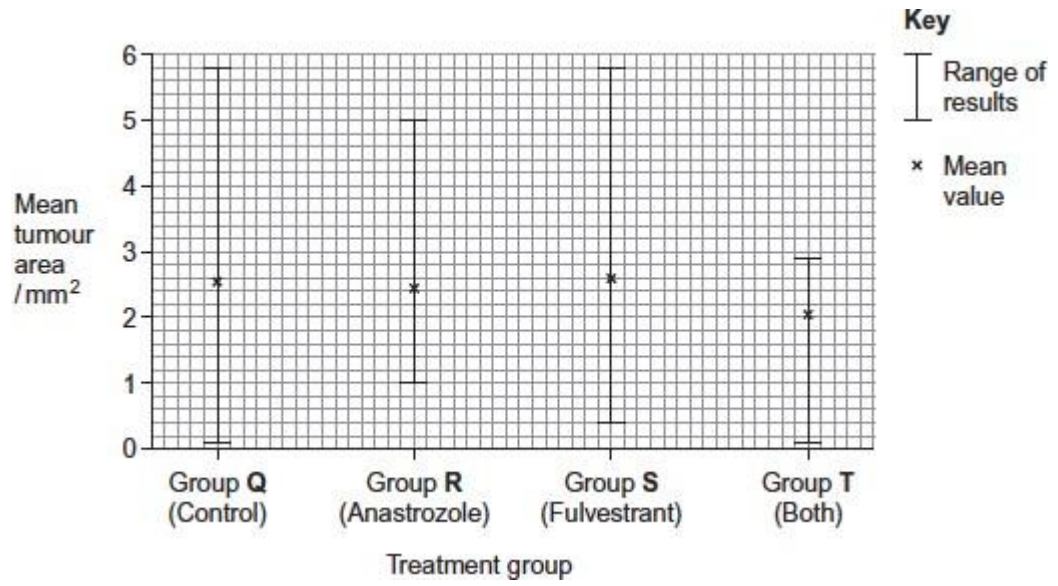


Figure 2



(c) The scientists concluded that both drugs should be used together to reduce the risk of lungcancer in women exposed to tobacco products.

Do you agree? Explain your answer.

(5)

(d) The scientists used tumour area as an indicator of tumour size.

Explain why tumour area may **not** be the best indicator of tumour size and suggest a more reliable measurement.

(2)

(e) The scientists repeated the investigation but this time they did not give the drugs until week9.

Suggest why they gave the drugs at week 9, rather than at week 5.

(2)

(f) Another group of scientists is currently using these drugs in human trials. However, thecontrol group is **not** being given a placebo.

Suggest why a placebo is **not** being given and what is being given to this group instead.

(2)

(Total 15 marks)

5 (a) Explain how the structure of DNA is related to its functions.

(Extra space) _____

(6)

Scientists investigated three genes, **C**, **D** and **E**, involved in controlling cell division.
They studied the effect of mutations in these genes on the risk of developing lung cancer.

The scientists analysed genes **C**, **D** and **E** from healthy people and people with lung cancer.

- If a person had a normal allele for a gene, they used the symbol N.
- If a person had two mutant alleles for a gene, they used the symbol M.

They used their data to calculate the risk of developing lung cancer for people with different combinations of N and M alleles of the genes. A risk value of 1.00 indicates no increased risk. The following table shows the scientists' results.

Gene C	Gene D	Gene E	Risk of developing lung cancer
N	N	N	1.00
M	N	N	1.30
N	N	M	1.78
N	M	N	1.45

N = at least one copy of the normal allele is present
M = two copies of the mutant allele are present

(b) What do these data suggest about the relative importance of the mutant alleles of genes **C**, **D** and **E** on **increasing** the risk of developing lung cancer? Explain your answer.

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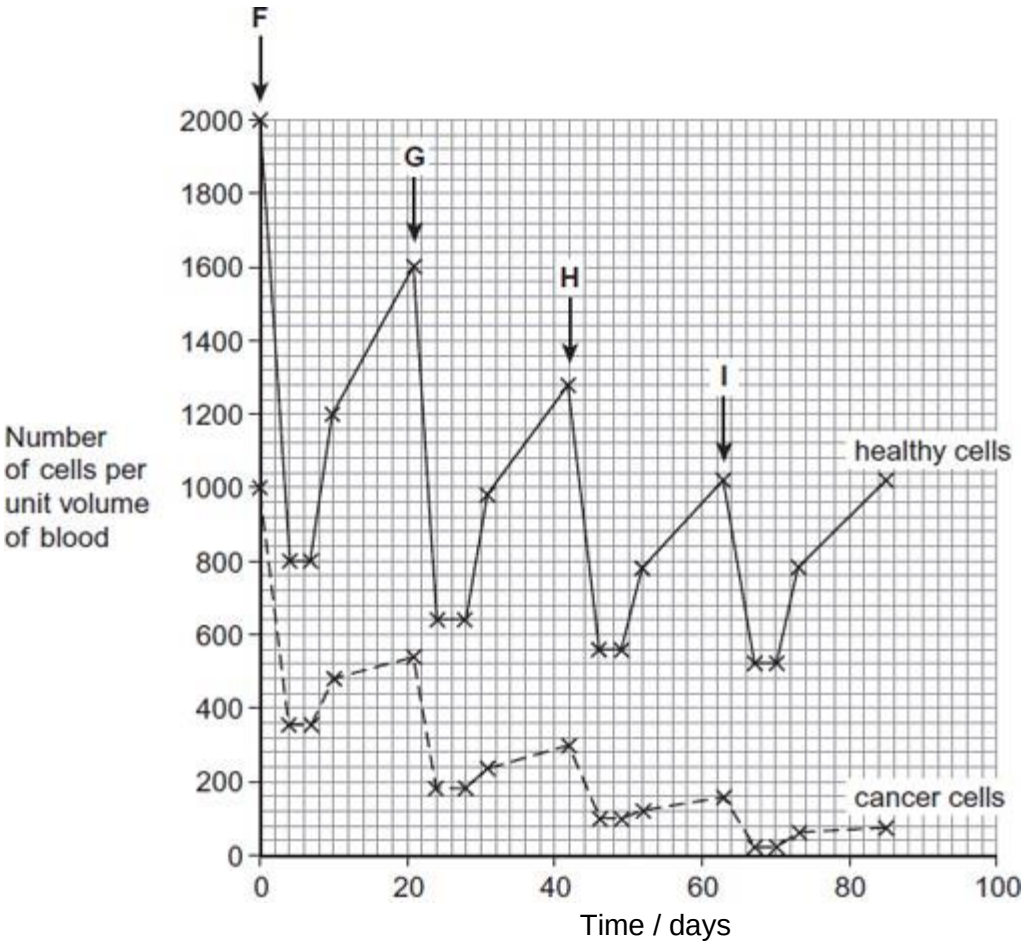
(3)

Chemotherapy is the use of a drug to treat cancer. The drug kills dividing cells.
The figure below shows the number of healthy cells and cancer cells in the blood of a patient receiving chemotherapy. The arrows labelled **F** to **I** show when the drug was given to the patient.



(c) Calculate the rate at which healthy cells were killed between days 42 and 46.

_____ cells killed per unit volume of blood per day

(1)

(d) Describe similarities and differences in the response of healthy cells and cancer cells to thedrug between times **F** and **G**.

_____

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(Extra space) _____

(3)

(e) More cancer cells could be destroyed if the drug was given more frequently.

Suggest why the drug was **not** given more frequently.

(2)

(Total 15 marks)

6 (a) (i) A mutation of a tumour suppressor gene can result in the formation of a tumour.

Explain how.

(2)

(ii) Not all mutations result in a change to the amino acid sequence of the encoded polypeptide.

Explain why.

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_____

(1)

(b) Some cancer cells have a receptor protein in their cell-surface membrane that binds to ahormone called **growth factor**. This stimulates the cancer cells to divide.

Scientists have produced a monoclonal antibody that stops this stimulation.

Use your knowledge of monoclonal antibodies to suggest how this antibody stops the growth of a tumour.

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[Extra space] _____

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(3)

(Total 6 marks)

7 Scientists investigated the effect of drinking tea and coffee on reducing the risk of developing one type of brain cancer. The investigation involved 410 000 volunteers and was conducted in 10 European countries over a period of 8.5 years.

(a) (i) Apart from age, suggest **two** factors that the scientists should have considered when selecting volunteers for this trial.

1. _____

2. _____

(2)

(ii) Give **two** features of the design of this investigation that would ensure the reliability ofthe results obtained.

1. _____

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2. _____

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(2)

(b) The incidence for this type of brain cancer is 6 cases per 100 000 per year.
Use this information to calculate the expected number of volunteers developing this cancer during the 8.5 year period of this investigation. Show your working.

Answer_____

(2)

(c) In analysing the results of this investigation, the scientists took into account the age of thevolunteers. Suggest why.

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(1)

(d) During the investigation, the volunteers were asked to estimate the volume of tea and/orcoffee that they drank each day. The types of tea and coffee consumed in different countries varied. When the data from all the countries were collected there was a correlation between drinking more than 100 $cm^3$of tea or coffee each day and a reduced risk of developing this type of brain cancer.

Tea and coffee contain caffeine. A newspaper reported the results of this investigation under the headline 'Caffeine helps cut cancer risk'. Explain why scientists could **not** support this view solely on the basis of this investigation.

(Extra space)

(4)

(e) Another group of scientists investigated the effect of caffeine on blood flow to certain parts of the brain. Volunteers were given different concentrations of caffeine solution to drink. A control group was also set up.

(i) Describe how the control group should have been treated.

(2)

(ii) Volunteers who drank the same concentration of caffeine solution often had differentconcentrations of caffeine in their blood. Suggest **one** reason for the difference in concentration of caffeine in the blood of volunteers.

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(1)

(iii) The investigation showed that caffeine reduces the blood flow to certain parts of thebrain. Suggest **one** way in which this could lead to a reduced risk of brain cancers.

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(1)

(Total 15 marks)

8 Imatinib is a drug used to treat a type of cancer that affects white blood cells. Scientists investigated the rate of uptake of imatinib by white blood cells. They measured the rate of uptake at 4°C and at 37°C.
Their results are shown in the table.

	Mean rate of uptake of imatinib into cells / µg per million cells per hour	
Concentration of imatinib outside cells / µmol dm^{-3}	**4°C**	**37°C**
0.5	4.0	10.5
1.0	10.7	32.5
5.0	40.4	420.5
10.0	51.9	794.6
50.0	249.9	3156.1
100.0	606.9	3173.0

(a) The scientists measured the rate of uptake of imatinib in µg per million cells per hour.Explain the advantage of using this unit of rate in this investigation.

(2)

(b) Calculate the percentage increase in the mean rate of uptake of imatinib when thetemperature is increased from 4°C to 37°C at a concentration of imatinib outside the cells of 1.0 μmol dm^{-3}.

Give your answer to one decimal place.

Answer _____

(2)

(c) Imatinib is taken up by blood cells by active transport.

(i) Explain how the data for the two different temperatures support this statement.

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(2)

(ii) Explain how the data for concentrations of imatinib outside the blood cells at 50 and 100 μmol dm^{-3} at 37°C support the statement that imatinib is taken up by active transport.

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(2)

(Total 8 marks)

9 Taxol is a drug used to treat cancer. Research scientists investigated the effect of injecting taxol on the growth of tumours in mice. Some of the results are shown in **Figure 1**.

Figure 1

Number of days of treatment	Mean volume of tumour / mm^3	
	Control group	Group injected with taxol in saline
1	1	1
10	7	2
20	21	11
30	43	20
40	114	48
50	372	87

(a) Suggest how the scientists should have treated the control group.

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(2)

(b) Suggest and explain **two** factors which should be considered when deciding the number of mice to be used in this investigation.

1. _____

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2. _____

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(2)

(c) The scientists measured the volume of the tumours. Explain the advantage of using volumerather than length to measure the growth of tumours.

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(1)

(d) The scientists concluded that taxol was effective in reducing the growth rate of the tumoursover the 50 days of treatment. Use suitable calculations to support this conclusion.

(2)

(e) In cells, taxol disrupts spindle activity. Use this information to explain the results in thegroup that has been treated with taxol.

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(3)

(f) The research scientists then investigated the effect of a drug called OGF on the growth oftumours in mice. OGF and taxol were injected into different mice as separate treatments or as a combined treatment. **Figure 2** and **Figure 3** show the results from this second investigation.

Figure 2

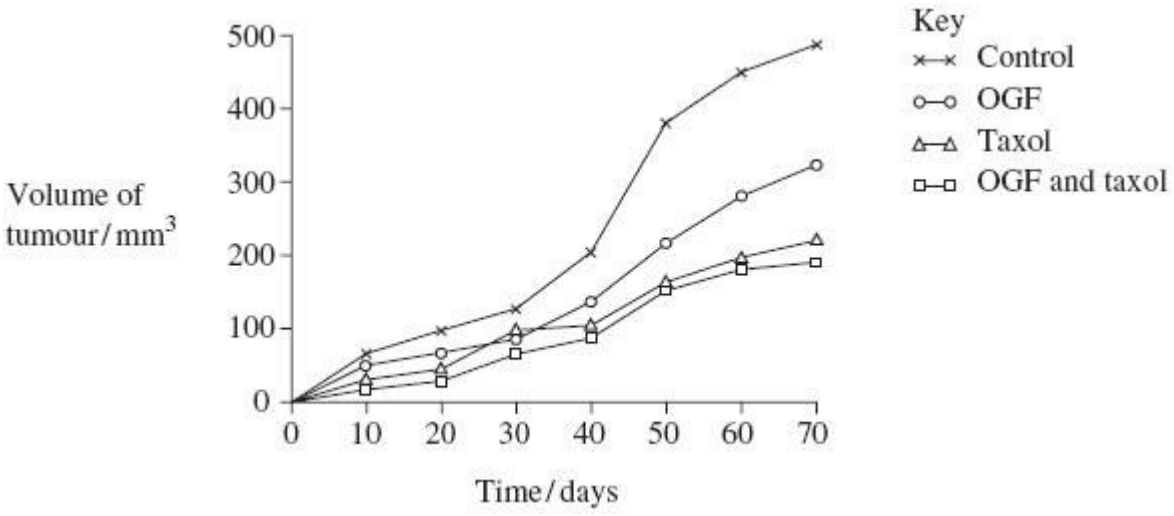

Figure 3

Treatment	Mean volume of tumour following 70 days treatment /mm^3 (± standard deviation)
OGF	322 (± 28.3)
Taxol	207 (± 22.5)
OGF and taxol	190 (± 25.7)
Control	488 (± 32.4)

(i) What information does standard deviation give about the volume of the tumours inthis investigation?

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(1)

(ii) Use **Figure 2** and **Figure 3** to evaluate the effectiveness of the two drugs when they are used separately and as a combined treatment.

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(4)

(Total 15 marks)

10 SCID is a severe inherited disease. People who are affected have no immunity. Doctors carried out a trial using gene therapy to treat children with SCID. The doctors who carried out the trial obtained stem cells from each child's umbilical cord.

(a) Give **two** characteristic features of stem cells.

1. _____

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2. _____

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(2)

The doctors mixed the stem cells with viruses. The viruses had been genetically modified to contain alleles of a gene producing full immunity. The doctors then injected this mixture into the child's bone marrow.

The viruses that the doctors used had RNA as their genetic material. When these viruses infect cells, they pass their RNA and two viral enzymes into the host cells.

(b) One of the viral enzymes makes a DNA copy of the virus RNA. Name this enzyme.

_____

(1)

The other viral enzyme is called integrase. Integrase inserts the DNA copy anywhere in the DNA of the host cell. It may even insert the DNA copy in one of the host cell's genes.

(c) (i) The insertion of the DNA copy in one of the host cell's genes may cause the cell tomake a non-functional protein. Explain how.

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(2)

(ii) Some of the children in the trial developed cancer. How might the insertion of the DNA have caused cancer?

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(2)

(d) Five out of the 20 children in the trial developed cancer. Although the cancer was treatedsuccessfully, the doctors decided to stop the trial in its early stages. They then reviewed the situation and decided to continue. Do you agree with their decision to continue? Explain your answer.

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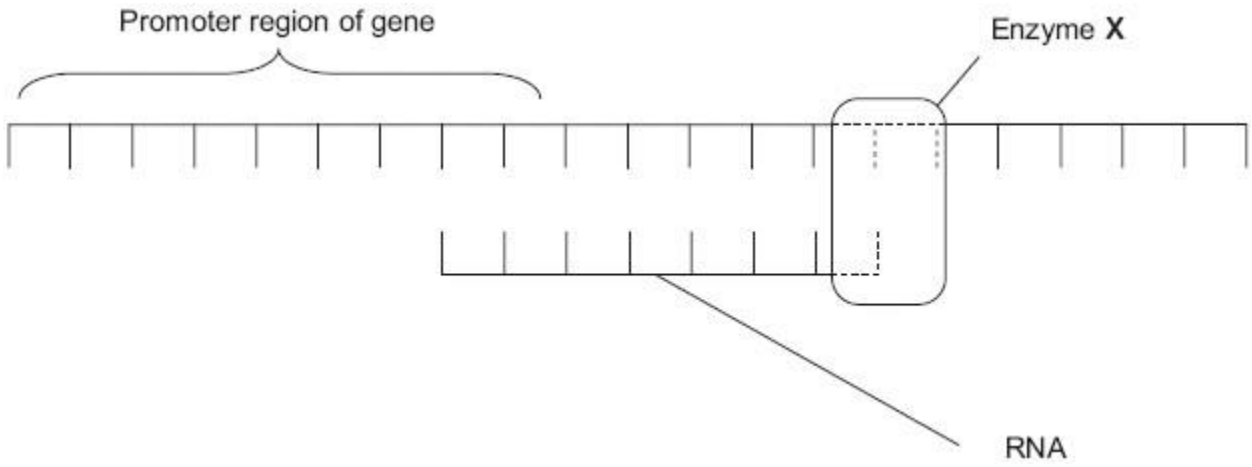
(2)

(Total 9 marks)

Figure 1 shows part of a gene that is being transcribed.

11

Figure 1



(a) Name enzyme **X**.

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(1)

(b) (i) Oestrogen is a hormone that affects transcription. It forms a complex with a receptorin the cytoplasm of target cells. Explain how an activated oestrogen receptor affects the target cell.

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(2)

(ii) Oestrogen only affects target cells. Explain why oestrogen does not affect other cells in the body.

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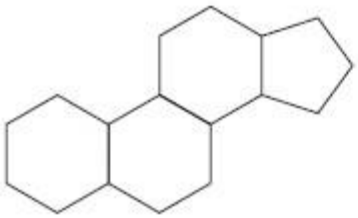
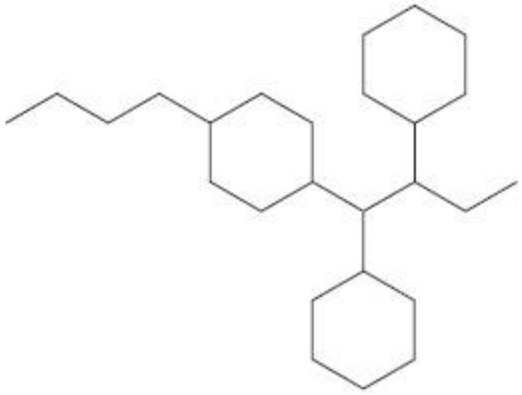
(1)

(c) Some breast tumours are stimulated to grow by oestrogen. Tamoxifen is used to treat these breast tumours. In the liver, tamoxifen is converted into an active substance called endoxifen. **Figure 2** shows a molecule of oestrogen and a molecule of endoxifen. **Figure 2**

Oestrogen



Endoxifen



Use **Figure 2** to suggest how endoxifen reduces the growth rate of these breast tumours.

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(2)

(Total 6 marks)

Essay

12

You should write your essay in continuous prose.

Your essay will be marked for its scientific accuracy.

It will also be marked for your selection of relevant material from different parts of the specification and for the quality of your written communication.

The maximum number of marks that can be awarded is

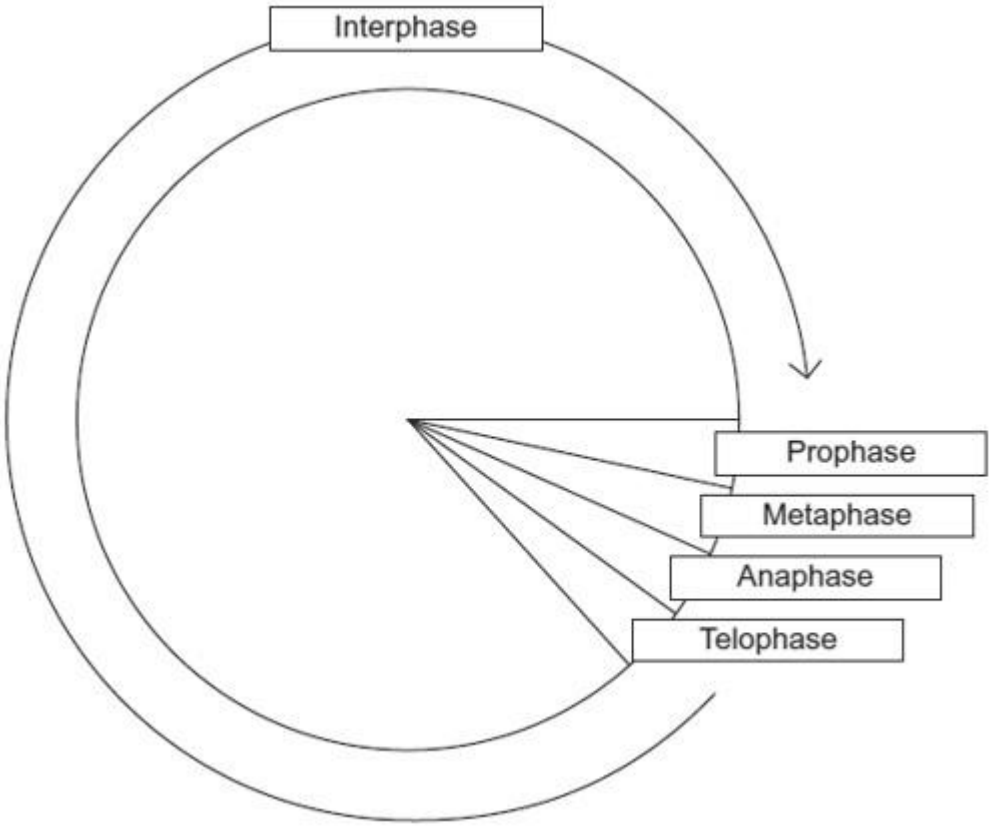
Scientific	16
Breadth of knowledge	3
Relevance	3
Quality of written communication	3

Write an essay on the following topic:

Using DNA in science and technology

(Total 25 marks)

13 The diagram shows a cell cycle.



(a) In prophase of mitosis, the chromosomes become visible. Describe what happens in

(i) metaphase

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(2)

(ii) anaphase.

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(2)

(b) (i) Cells lining the human intestine complete the cell cycle in a short time. Explain theadvantage of these cells completing the cell cycle in a short time.

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(1)

(ii) The time required for a cell to complete the cell cycle was 4 hours 18 minutes.

Calculate the time required in minutes for this cell to multiply to produce eight cells. Show your working.

Answer _____

(2)

(c) Mikanolide is a drug that inhibits the enzyme DNA polymerase. Explain why this drug may be effective against some types of cancer.

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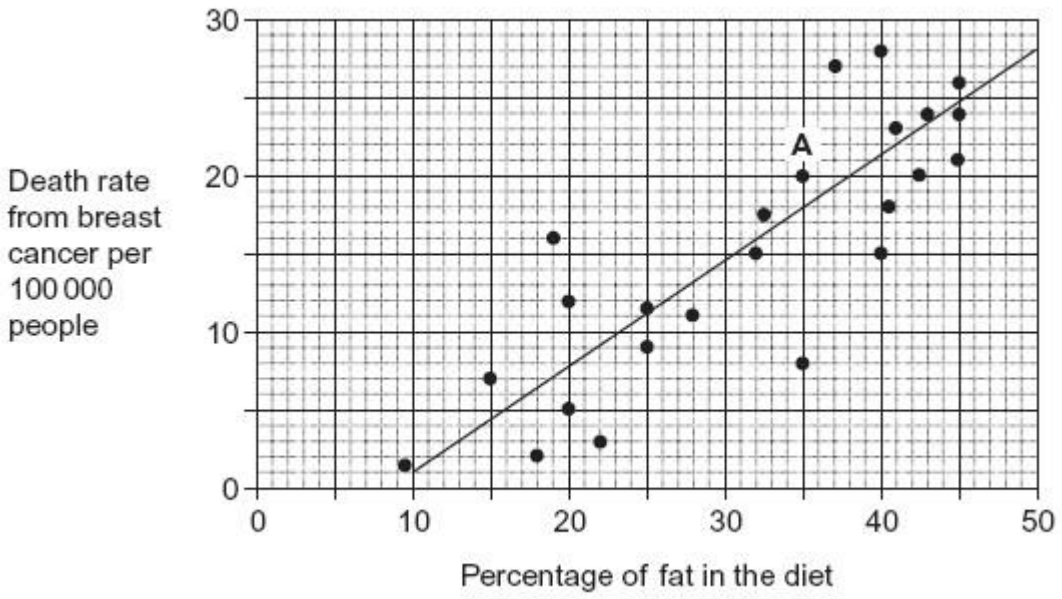
(2)

(Total 9 marks)

14 Scientists investigated the relationship between the percentage of fat in the diet and the death rate from breast cancer in 24 different countries. They plotted the data from each country on the graph below.



(a) Describe the information given by point **A** on the graph.

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(1)

(b) Describe how the scientists calculated the death rate from breast cancer for each country.

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(1)

(c) Some people have used the graph to conclude that a high percentage of fat in the diet causes breast cancer. Evaluate this conclusion.

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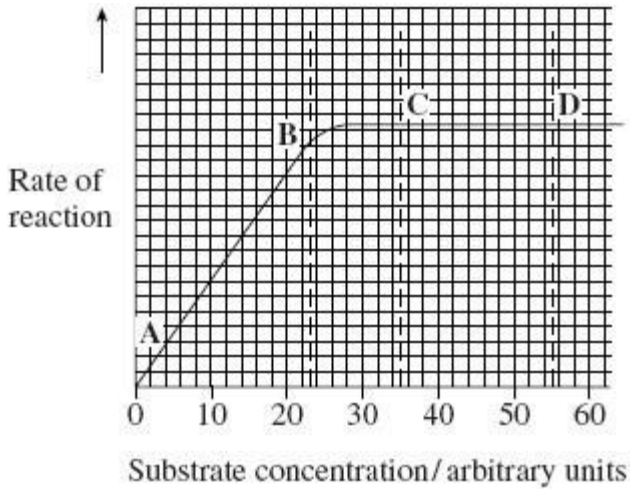
(3)

(Total 5 marks)

15 The graph shows the effect of substrate concentration on the rate of an enzyme-controlled reaction.



(a) (i) Describe what the graph shows about the effect of substrate concentration on therate of this enzyme-controlled reaction.

(2)

(ii) What limits the rate of this reaction between points **A** and **B**? Give the evidence from the graph for this.

(2)

(iii) Suggest a reason for the shape of the curve between points **C** and **D**.

(1)

(b) Sketch a curve on the graph to show the rate of this reaction in the presence of acompetitive inhibitor.

(1)

(c) Methotrexate is a drug used in the treatment of cancer. It is a competitive inhibitor andaffects the enzyme folate reductase.

(i) Explain how the drug lowers the rate of reaction controlled by folate reductase.

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(2)

(ii) Methotrexate only affects the rate of the reaction controlled by folate reductase.

Explain why this drug does not affect other enzymes.

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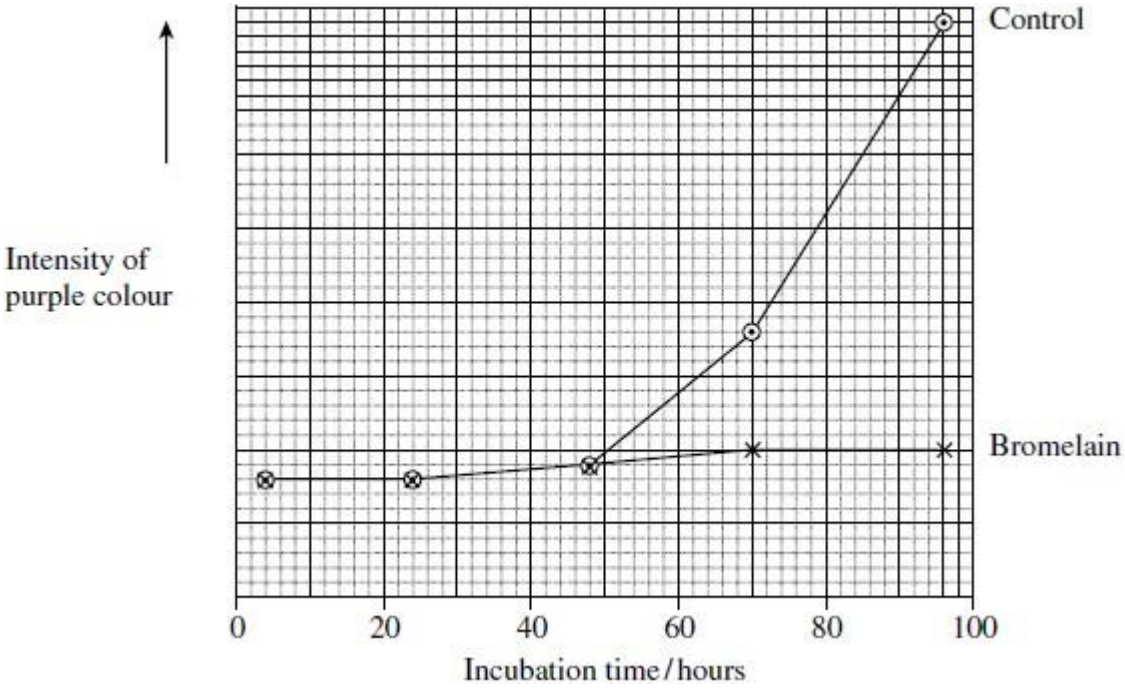
(1)

(Total 9 marks)

16 Scientists investigated the effect of bromelain on cancer cells. They took cells from skin cancers in mice and added them to a liquid growth medium in two dishes.

Four hours later they added a solution of bromelain to one of the dishes. They left the other dish as a control. They also added a substance to both dishes that is turned purple by respiring cells.

Both dishes were placed in an incubator. The scientists measured the intensity of the purple colour at intervals over a period of 100 hours.



(a) The scientists put the same number of skin tumour cells in each dish at the start of thisinvestigation. Explain why it was important to put the same number of cells in each dish.

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(1)

(b) The scientists concluded that bromelain did not kill cancer cells but stopped them dividing.Does the graph support this conclusion? Explain your answer.

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(2)

(c) An article in a newspaper claimed that these data show that bromelain can be used to treatcancer.

Give **three** reasons why we should be careful about accepting this claim.

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2. _____

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3. _____

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(3)

(d) The rate of cell division is important in investigations into cancer. Suggest why.

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(2)

(e) Scientists have investigated the effects of bromelain on cancer growth in humans. Suggestwhy they gave bromelain in addition to, rather than instead of, the usual treatment.

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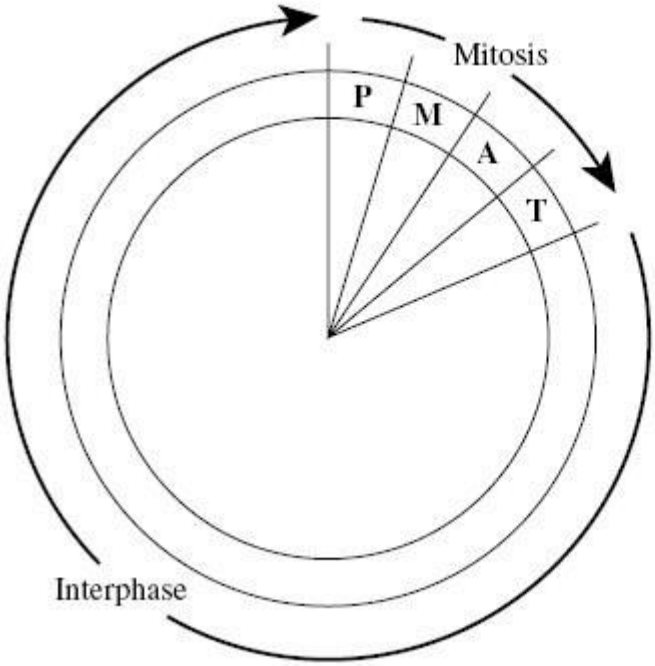
(2)

(Total 10 marks)

17 The diagram shows a cell cycle.



Key

P prophase

M metaphase

A anaphase

T telophase

(a) The table shows the number of chromosomes and the mass of DNA in different nuclei.

All the nuclei come from the same animal. Complete this table.

Nucleus	Number of chromosomes	Mass of DNA / arbitrary units
At prophase of mitosis	26	60
At telophase of mitosis		
From a sperm cell		

(4)

(b) If the DNA of the cell is damaged, a protein called p53 stops the cell cycle.

Mutation in the gene for p53 could cause cancer to develop. Explain how.

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(3)

(c) Drugs are used to treat cancer. At what phase in the cell cycle would each of the followingdrugs act?

(i) A drug that prevents DNA replication

_____

(1)

(ii) A drug that prevents spindle fibres shortening

_____

(1)

(Total 9 marks)

18 Read the following passage.

The idea that bacteria could be used as a cancer treatment originated over 100 years ago. A doctor noticed that some cancer patients with bacterial infections showed signs of recovery from the cancer. Attempts to use the bacteria as a treatment were disappointing, however. Experiments showed that the bacteria made an impressive onslaught on tumours, but a ring of cancerous tissue around the edge usually survived.

Bacteria are once again being used in the war on cancer. Scientists have genetically engineered a harmless strain of *Clostridium* to carry the gene for an enzyme. This enzyme converts a harmless "prodrug" into an active drug which acts as a powerful toxin. In people, this strain of *Clostridium* will only grow in tumours. Scientists hope that when they inject the prodrug into a cancer patient's blood, the bacteria will convert it into an active drug. This will destroy tumours from the inside, leaving healthy tissues unharmed.

The idea of converting a harmless prodrug into an active drug that only kills cancer cells is not new. Apart from the use of genetically modified *Clostridium*, other methods have been tried. One of these involved attaching an enzyme to an antibody that binds only to cancer cells. This enzyme then activates the drug. Unfortunately, different types of cancer require different antibodies, making the treatment expensive to develop. Scientists hope their bacterial approach will offer a way of delivering the enzymes to any cancer cell.

(a) Describe how scientists could genetically engineer *Clostridium* bacteria to produce the enzyme which activates the prodrug. (lines 7-8)

(6)

(b) Explain why it is important to destroy all the cancer cells in a tumour.

(2)

(c) Explain how the use of antibodies (lines 16-17) results in a drug only killing cancer cells.

_____

_____

(3)

(d) Cancer drugs usually interfere with DNA replication. Use this information to explain why thecancer drugs are administered as prodrugs and not the active form.

_____

_____

_____

_____

_____

_____

_____

_____

(4)

(Total 15 marks)

19

(a) Some tumours are benign and some are malignant.

(i) Give **one** way in which a benign tumour differs from a malignant tumour.

_____

(1)

(ii) Describe **two** ways in which both types of tumour may cause harm to the body.

1. _____

_____

2. _____

_____

(2)

(b) (i) Explain the link between sunbathing and skin cancer.

_____

_____

_____

_____

(2)

(ii) Suggest why fair-skinned people are at a greater risk of skin cancer thandark-skinned people when sunbathing.

_____

_____

(1)

(iii) Suggest why people with a family history of cancer are at a greater risk of cancerthan those with no family history of cancer.

_____

_____

(1)

(Total 7 marks)

Read the following passage.

20

Soon a single drop of blood might be enough to reveal, at a very early stage, if a patient has cancer. It could also tell us what type of cancer it is and whether it is treatable. Fragments of DNA from body cells are present in blood plasma. Some of these fragments may be from cancer cells. The fragments can be detected by a new test in which a test strip containing 5 nucleic acid binds to sections of altered DNA.

Other cancer-detecting techniques involve removing a tissue sample from a patient. The tissue sample is used to obtain mRNA. By examining the mRNA, scientists can discover whether cancer is present.

Use information from the passage and your own knowledge to answer the questions.

(a) Describe how altered DNA may lead to cancer.

(6)

(b) Explain why fragments of DNA from cancer cells may be present in blood plasma(lines 3-4).

(2)

(c) Explain why the nucleic acid on the test strip will only bind to altered DNA (lines 4-5).

(2)

(d) This test strip will allow cancers to be detected at a very early stage. Explain why cancer ismore likely to be treated successfully if the disease is detected at a very early stage.

(2)

(e) Explain how examining mRNA (line 7) enables scientists to discover whether cancer ispresent.

(3)

(Total 15 marks)

Lung cancer, chronic bronchitis and coronary heart disease (CHD) are associated with smoking.

21

Tables 1 and **2** give the total numbers of deaths from these diseases in the UK in 1974.

Table 1 Men

Age/years	Number of deaths (in thousands)		
	lung cancer	chronic bronchitis	coronary heart disease
35 - 64	11.5	4.2	31.7
65 - 74	12.6	8.5	33.3
75+	5.8	8.1	29.1
Total (35 - 75+)	29.9	20.8	94.1

Table 2 Women

Age/years	Number of deaths (in thousands)		
	lung cancer	chronic bronchitis	coronary heart disease
35 – 64	3.2	1.3	8.4
65 – 74	2.6	1.9	18.2
75+	1.8	3.5	42.3
Total (35 – 75+)	7.6	6.7	68.9

(i) Using an example from the tables, explain why it is useful to give data for men and womenseparately.

_____

_____

_____

(2)

(ii) Data like these are often given as percentages of people dying from each cause.

Explain the advantage of giving these data as percentages.

_____

_____

_____

_____

(2)

(Total 4 marks)

22 One hypothesis for the cause of cancer of the colon (large intestine) is that *Clostridium* bacteria present in the gut can convert bile steroids into cancer-causing substances.

S (a) Explain the presence of bile in the colon.

_____

_____

_____

_____

(2)

(b) The concentrations of bile steroids and numbers of *Clostridium* bacteria were measured in people with colon cancer and in controls without colon cancer. The table shows the results.

Concentration of bile steroids	Number of *Clostridium* bacteria	Percentage of cancer patients	Percentage of controls	P
high	high	76	9	<0.01
high	low	13	8	<0.01
low	high	7	34	<0.01
low	low	4	49	<0.01

A statistical test showed there was a significant difference between the cancer patients and the controls in each of the four categories.

(i) Explain how the results could be used to support the hypothesis that *Clostridium* bacteria convert bile steroids into substances which cause colon cancer.

_____

_____

_____

_____

(2)

(ii) Explain how the results indicate that other factors may be involved in causing coloncancer.

_____

_____

(1)

S (c) Human cells contain genes that control their growth and division. One of these genes codes for a protein that prevents cell division. The substances formed from bile steroids by *Clostridium* bacteria may cause gene mutation. Describe and explain how these substances could cause colon cancer.

_____

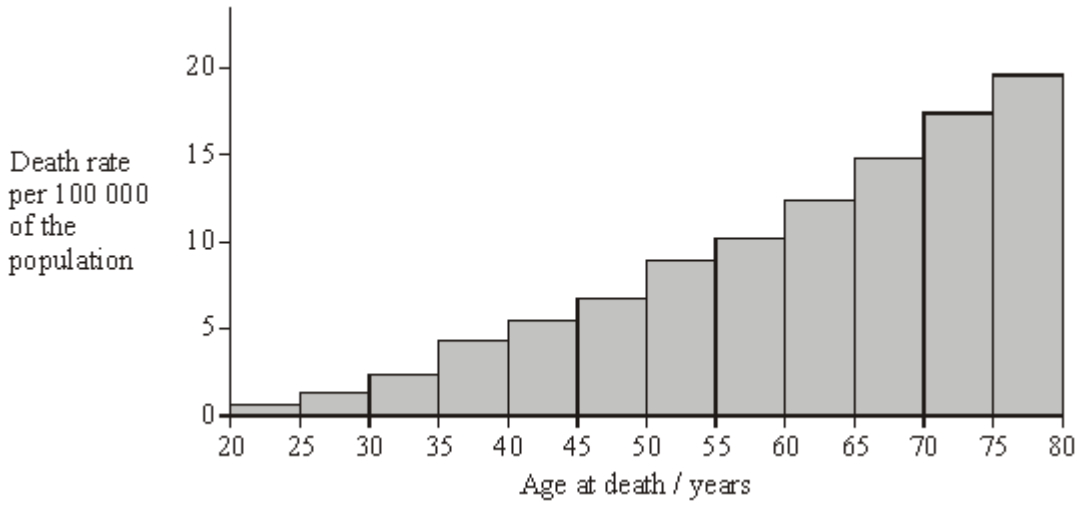
_____

_____

(4)

(Total 9 marks)

23 The death rate from malignant skin tumours was investigated in the USA. The graph shows the results for fair-skinned men in different age groups.



(a) Describe what is meant by a *malignant tumour*.

(3)

(b) Give **one** reason for the change in death rate from malignant skin tumours with increasing age.

_____

_____

(1)

(c) The data for fair-skinned and dark-skinned people were collected separately.Explain why skin colour was a factor likely to affect the death rate.

_____

_____

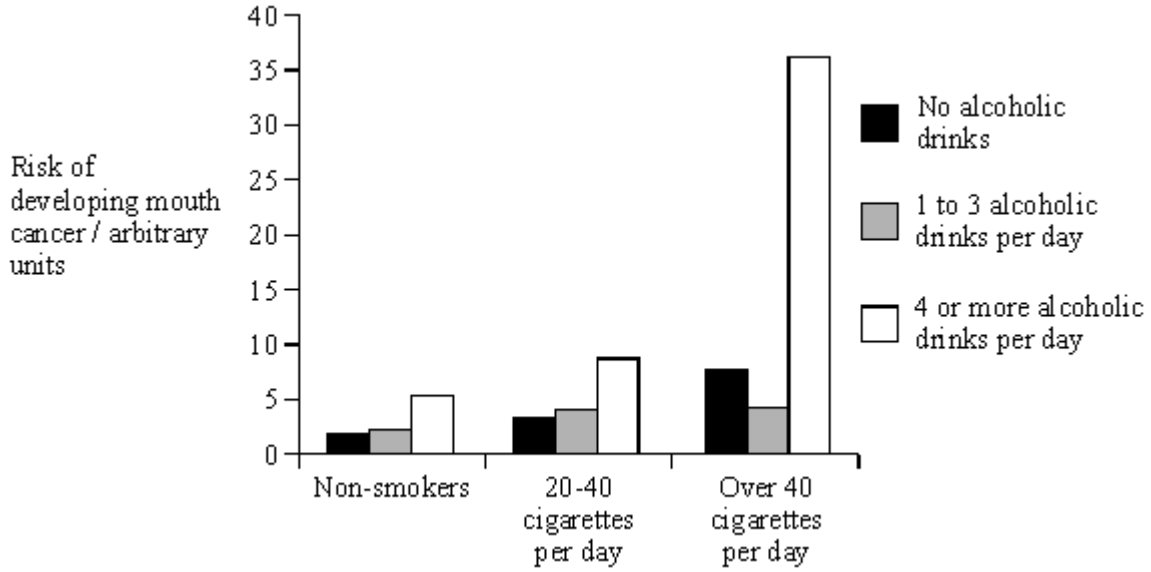
_____

_____

(2)

(Total 6 marks)

24 The bar chart shows the effects of smoking and alcoholic drinks on the risk of developing mouth cancer.



(i) Describe the effects of smoking and drinking on the risk of developing mouth cancer.

_____

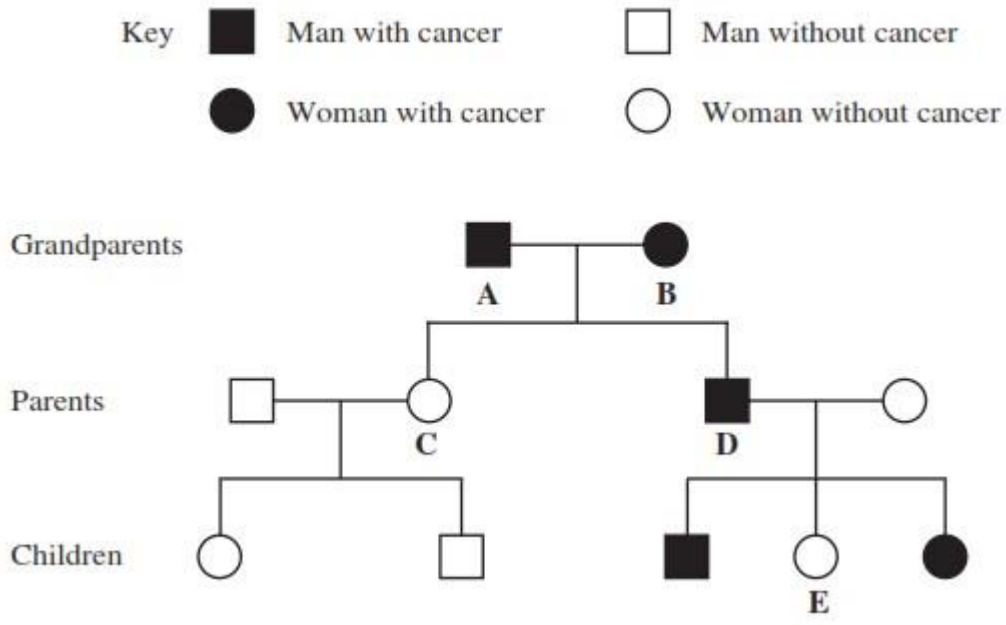
(3)

(ii) Suggest **one** reason why people who neither drink nor smoke sometimes develop mouth cancer.

(1)

(Total 4 marks)

25 Li-Fraumeni syndrome is a rare inherited condition. It makes someone much more likely to develop cancer at an early age. The diagram shows part of the family history of a family affected by Li-Fraumeni syndrome. Li-Fraumeni syndrome is caused by the dominant allele of a gene. The gene is not sex-linked.



The grandparents, **A** and **B**, had two children, girl **C** and boy **D**. Explain how the phenotypes of these children provide evidence that Li-Fraumeni syndrome is

(a) caused by a dominant allele

(2)

(b) **not** sex-linked.

(2)

(c) This family's history of cancer was investigated when person **E** asked for genetic counselling. At the time she was 25 years old. What advice could a genetic counsellor give her about her probability of developing cancer?

(2)

(d) Li-Fraumeni syndrome is caused by a mutation affecting a tumour suppressor gene calledTP53. This gene codes for a protein that initiates the death of cells where damaged DNA cannot be repaired. The mutated TP53 gene leads to the production of a non-functional protein. Suggest how the non-functional protein may lead to cancer.

_____

_____

_____

_____

(Extra space) _____

_____

_____

(3)

(Total 9 marks)

26 Scientists found a correlation between prostate cancer and exposure to cadmium ions.

The scientists investigated the effects of cadmium ions on cells from a human prostate gland. They grew a culture of these cells in liquid growth medium and removed samples at intervals.

For each sample they measured

- how much DNA was not methylated,
- the activity of the enzyme methyltransferase.

Methyltransferase is an enzyme that adds methyl groups to some of the bases in DNA. The addition of a methyl group is called methylation.

(a) The scientists set up another culture as a control.

Describe how the scientists would have set up a control experiment for this investigation.

_____

_____

_____

_____

(2)

(b) **Figures 1** and **2** show the scientists' results.

Figure 1

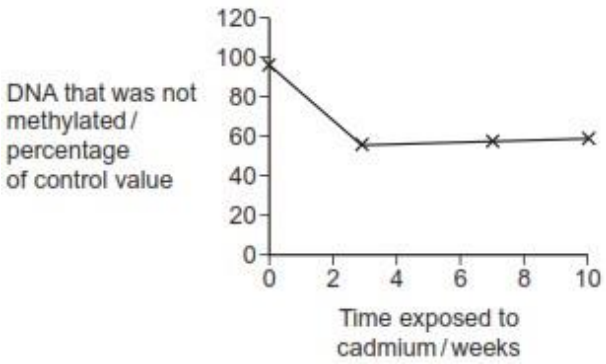
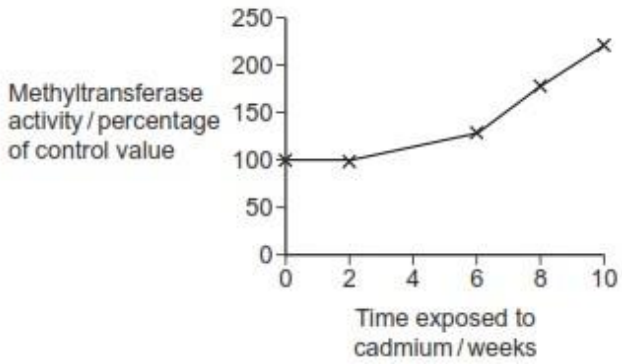


Figure 2



(i) The scientists expressed their results as percentages of the control values.Suggest why.

_____

_____

(1)

(ii) Use information from **Figure 1** to describe how exposure to cadmium ions affected the methylation of DNA.

_____

_____

(1)

(iii) Use information from **Figure 2** to suggest what caused the change to the DNA shown in **Figure 1**.

_____

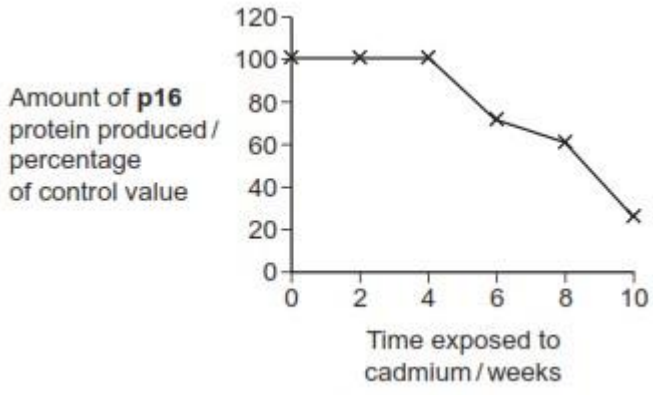
_____

(1)

(c) Prostate gland cells contain a tumour suppressor gene called **p16**.
During the investigation, the scientists also measured the amount of **p16** protein produced.

Figure 3 shows their results.

Figure 3



The scientists found that the promoter DNA of the **p16** gene had become methylated. The promoter is the sequence of bases where the enzyme RNA-polymerase binds to a DNA molecule.

Explain how methylation of the promoter sequence of the **p16** gene could cause the changes shown in **Figure 3**.

_____

_____

_____

_____

(Extra space) _____

_____

(2)

(d) Each week of the investigation, the scientists took samples of the cadmium-treated prostate cells from the laboratory cultures. They injected these cells into mice and monitored the mice for the growth of tumours.

It was only the samples taken in the tenth week that caused tumours to begin to grow in the mice.

Use information from **Figures 1, 2** and **3** to suggest why.

_____

_____

_____

_____

_____

_____

_____

_____

(Extra space) _____

_____

_____

_____

(4)

(Total 11 marks)

Mark schemes

(a) 1. Methylation prevents transcription of gene;

1

2. Protein not produced that prevents cell division / causes cell death / apoptosis;3.
No control of mitosis.

3

(b) 1. Scatter graph;
2. Fat on *x* axis and death rate on *y* axis;
3. (Because) looking at relationship between two discrete / independent variables.

3

(c) 1. (Trend) shows positive correlation / shows the more fat in diet, the higher deathrate from breast cancer;
2. But number of points off line / anomalies.

2

[8] (a) 1. Rank all STs in ascending order;

2

2. Find value with same number (of people) above and below.

Accept find middle value

2

(b) Not ethical to fail to treat cancer.

1

(c) Yes since with ipilimumab:

1. Median ST increased by 2.1 months;
2. Percentage of patients showing reduction in tumours increased from 10.3% to15.2%;

No because:

3. No standard errors shown / no (Student) t- test / no statistical test carried out;
4. (So) not able to tell if differences are (statistically) significant / due to chance(alone);
5. Improvement might only be evident in some patients / no improvement in somepatients;
6. Quality of (extra) time alive not reported;

If answers relate only to 'Yes' or 'No', award 2 marks max

4 max

(d) 1. Faulty protein recognised as an antigen / as a 'foreign' protein;
2. T cells will bind to faulty protein / to (this) 'foreign' protein;
3. (Sensitised) T cells will stimulate clonal selection of B cells;4.
(Resulting in) release of antibodies against faulty protein.

3 max

[10]

3

(a) 1. Binding (of interferon gamma) changes shape/tertiary structure of receptor (protein);

2. This activates/switches on the enzyme;
3. Use of ATP (to phosphorylate STAT1);

1. Accept reference to second messenger mechanism/process3.

Context is important

2 max

(b) 1. Phosphorylated STAT1;
2. IRF (protein);

Accept in either order

1. Must be phosphorylated but accept STAT1P

2. Ignore references to phosphorylated

2

(c) 1. Causes more helper T cells to form;
2. (So) more interferon (gamma) production (by helper T cells);

1. and 2. require idea of more

2

(d) 1. (Tumour suppressor gene) slows cell division/causes death ofdamaged/tumour/cancer cells;
2. *IRF* gene leads to formation of IRF (protein) that binds to gene B;
3. (Gene B protein) causes death of damaged/mutated cells ORslows division;

2. 'It' means IRF gene

3. Context is important

*3. If clearly stated **and** includes the protein, scores 2 marks because it subsumes point 1*

3

[9]

4

(a) 1. Removes (main / largest) source of oestrogen / (different) mice produce different amounts of oestrogen;

Accept: so oestrogen from ovaries not a confounding variable − idea of.

2. (Allows) oestrogen to be controlled / oestrogen to be made by aromatase only /only oestrogen made in lungs to be involved.

Reject: references to injection of aromatase.

2

(b) 1. (Anastrozole) prevents / reduces oestrogen production;

2. (Fulvestrant) stops remaining oestrogen binding / less oestrogen binds toreceptors.

Note: brackets around drug names.

2

(c) (Yes for Group T)

1. Least tumours per animal (from fig. 1);

Accept: 'mean values' for tumour area.

2. Lowest (mean) tumour area / size (from fig. 2);
3. Lowest top of range;

(But)

4. Means (tumour area) are similar;

Where candidates confuse range and standard deviation, do not give credit.

5. Ranges overlap / share values so differences may not be real / treatments may be just effective in reducing tumour;

Ignore significance

6. Range affected by outliers / SD's would be better;
7. Done on mice / not done on women / humans;
8. Only 10 mice used per group / small sample size so may not be representative / reliable;
9. Might be side effects;
10. Only did for 15 weeks so maximum effect of drugs may not have been seen.

5 max

(d) 1. Tumours may be different depths / area does not take depth into account /tumours are 3-D / are not 2-D;

Neutral: different sizes

Accept: height / thickness for depth

2. (Measure) tumour volume / mass / weight.

2

(e) 1. Allows tumours to grow / develop / form;

Neutral: gives drug more time to work.

2. (So) can investigate treatment rather than prevention (of tumours) / when tumour / cancer is more advanced.

Accept: to see whether it can destroy / treat / stop growth of a tumour (that already exists) / to allow / assess treatment of a tumour

2

(f) 1. Unethical (not to treat patients) / may increase probability of patients dying /getting more ill;

Reject: references to giving people tumours

2. Use normal cancer drugs / treatment.

Accept: named type of cancer treatment, e.g. chemotherapy

2

[15]

5 (a) 1. Sugar-phosphate (backbone) / double stranded / helix **so** provides strength / stability / protects bases / protects hydrogen bonds;

Must be a direct link / obvious to get the mark

Neutral: reference to histones

2. Long / large molecule **so** can store lots of information;

3. Helix / coiled **so** compact;

Accept: can store in a small amount of space for 'compact'

4. Base sequence allows information to be stored / base sequence codes foramino acids / protein;

Accept: base sequence allows transcription

5. Double stranded **so** replication can occur semi-conservatively / strands can act as templates / complementary base pairing / A-T and G-C so accurate replication / identical copies can be made;

6. (Weak) hydrogen bonds **for** replication / unzipping / strand separation / many hydrogen bonds **so** stable / strong;

Accept: 'H-bonds' for 'hydrogen bonds'

6

(b) 1. (Mutation) in **E** produces highest risk / 1.78;

2. (Mutation) in **D** produces next highest risk / 1.45;

3. (Mutation) in **C** produces least risk / 1.30; *Must be stated directly and not implied*

E > ***D*** > ***C*** *= 3 marks*

Accept: values of 0.78, 0.45 and 0.30 for MP1, MP2 and MP3 respectively

If no mark is awarded, a principle mark can be given for the idea that all mutant alleles increase the risk

3

(c) **180**;

1

(d) **(Similarities):**

1. Same / similar pattern / both decrease, stay the same then increase;

2. Number of cells stays the same for same length of time;*Ignore: wrong days stated*

(Differences):

(Per unit volume of blood)

3. Greater / faster decrease in number of healthy cells / more healthy cells killed /healthy cells killed faster;

Accept: converse for cancer cells

Accept: greater <u>percentage</u> decrease in number of cancer cells / greater <u>proportion</u> of cancer cells killed

4. Greater / faster increase in number of healthy cells / more healthy cellsreplaced / divide / healthy cells replaced / divide faster;

Accept: converse for cancer cells

*For **differences**, statements made must be comparative*

3 max

(e) 1. More / too many healthy cells killed;

2. (So) will take time to replace / increase in number;*Neutral: will take time to 'repair'*

3. Person may die / have side effects;

2 max

[15]

6 (a) (i) 1. (Tumour suppressor) gene inactivated / not able to control / slow down cell division;

Ignore: references to growth

2. Rate of cell division too fast / out of control.

1 and 2 Accept: mitosis

1 and 2 Reject: meiosis

2

(ii) 1. (Genetic) code degenerate;

Accept: codon for triplet

Accept description of degenerate code, e.g. another triplet codes for the same amino acid

2. Mutation in intron.

Accept: mutation in non-coding DNA

1 max

(b) 1. Antibody has specific tertiary structure / binding site / variable region;

Do not accept explanations involving undefined antigen

2. Complementary (shape / fit) to receptor protein / GF / binds to receptor protein /to GF;

Ignore: same shape as receptor protein / GF

3. Prevents GF binding (to receptor).

3

[6] (a) (i) 1. Sex;

7

2. Lifestyle;
   Stress, smoking, diet etc are examples of lifestyle.

3. Body mass;
   3. Allow weight for mark point 3.

4. Health;
   Reject: height.

5. Ethnicity;

6. Genetic factors / family history;

2 max

(ii) 1. Large sample / number / 410 000;
   Reject: random

2. Long time period / 8.5 / many years;

3. Different countries / more than one country;

2

(b) Correct answer of 209 / 209.1 = 2 marks;
   Answer of 210 = one mark

Incorrect answer but multiplies by 8.5 = 1 mark;

2

(c) Age affects risk of cancer;
   Must relate to cancer not just to illness

1

(d) 1. Correlation does not mean causal relationship;
1. Reject casual for point 1.
   Reference to 'due to other factors' on its own is not enough for a mark

2. Tea / coffee contains other substances / different amounts of caffeine /estimated intake (of tea / coffee);

3. No control group;

4. Only one type of cancer studied / further studies required / only oneinvestigation / study / group;

4

(e) (i) 1. Treated the same;
2. Accept decaffeinated

2. No caffeine;
2. Reject placebo.

2

(ii) 1. Absorb different amounts;
Reject: Different body masses

2. Broken down by enzymes / digested;

3. Different blood volumes;

4. Differences in metabolism;

5. Caffeine from a different source;

1 max

(iii) 1. Less oxygen / glucose to (cancer) cells;
'Reduces cell division' on its own should not be credited.

2. Less carcinogens;

3. Reduces spread of cancer (cells);

1 max

[15] (a) 1. To allow comparison;

8

2. Because different number of cells in samples / different times for incubation / numbers become easier to manipulate;

2

(b) 203.7(%);;
Allow 1 mark for 21.8 / 10.7
Allow 1 mark for correct answer (203.74) but not correctly to 1 dp
204 = 1 mark

2

(c) (i) 1. (At every concentration) uptake is faster at 37°C / at higher temperature;

2. Due to faster respiration / ATP production;

2

(ii) 1. Uptake at 37°C only small increase / levelling off / almost constant as carrier proteins full;

Accept 'no (significant) change'

Ignore use of numbers

2. Concentration of imatinib is not the limiting factor;

2

[8]

9 (a) Given only saline;

Otherwise treated exactly the same way;

2

(b) Ethical consideration, e.g., leads to death / suffering of mice;

Large number to improve reliability / reduce sampling error;

Number of mice related to cost / space available / animal husbandry;

2 max

(c) Vary in shape / do not grow uniformly;

Q *Allow descriptions of variation in shape.*

1

(d) 7.44 and 1.74;;

7.42 and 1.72;;

(Ratio) 4.28 : 1;;

(Ratio) 4.31 : 1;;

(Percentage decrease) 76.6%;;

(Percentage decrease) 76.8%;;

Any of the answers shown gain two marks.

An answer of 23.4% or 23.2%
Percentage decrease gains one mark.

Correct method of calculating rate / ratio / percentage increase with an incorrect answer gains one mark.

2 max

(e) Reference to Mitosis;

As chromosomes cannot attach (to spindle) / chromatids cannot separate (on spindle);

Q *Do not penalise confusion between chromosomes and chromatids in second marking point*

Cell division / cell cycle slows down;

Q *Mitosis slows down = 2 marks*

Q *Mitosis stopped = 1 mark*

Q *Mitosis must be spelt correctly*

3

(f) (i) (Degree of) spread / variation from the mean;

1

(ii) Both chemicals (on their own) slow down growth / are effective;

Taxol is more effective than OGF;

Combined treatment (seems) most effective;

SD overlap for OGF with taxol and taxol (on its own) so not conclusive / could be chance / both treatments could be equally effective;

Q *Ignore all references to significance*

4

[15] (a) Will replace themselves / keep dividing / replicate;

10

Undifferentiated / can differentiate / develop into other cells / totipotent / multipotent / pluripotent;

Accept tissues

2

(b) Reverse transcriptase;

Allow phonetic spelling

1

(c) (i) Alters base / nucleotide sequence / causes frame shift;

Different sequence of amino acids in polypeptide / protein / primary structure alters the tertiary structure;

Accept any reference, such as adding bases, to changing the base sequence of the gene. Reject deletion / substitution.
Idea of sequence essential so not makes different amino acids.

Accept answers involving stop / start codons and effect on protein.

2

(ii) Affects tumour suppressor gene;

Inactivates (tumour suppressor) gene;

Rate of cell division increased / tumour cells continue to divide;

Ignore answers relating to oncogenes. May gain third point.

2 max

(d) Yes
SCID patients unlikely to survive / quality of life poor unless treated;
Cancer that develops is treatable / only affects 25% / five children;

No
Risk of developing cancer is high / 25%;
Cancer may recur / may not be treated successfully in future / only short time scale so more may develop cancer;

No mark for yes or no. Marks are for supporting argument based on biological reasoning.
Accept any points

2 max

[9]

11 (a) RNA polymerase;

DNA polymerase is incorrect
Ignore references to RNA dependent or DNA dependent
Allow phonetic spelling

1

(b) (i) (Receptor / transcription factor) binds to promoter which stimulates RNApolymerase / enzyme X;

Transcribes gene / increase transcription;

2

(ii) Other cells do not have the / oestrogen / ERα receptors;

But do not accept receptors in general.

1

(c) Similar shape to oestrogen;

Binds receptor / prevents oestrogen binding;

Receptor not activated / will not attach to promoter / no transcription;

Accept alternative
Complementary to oestrogen;
Binds to oestrogen;
Will not fit receptor;

2 max

[6]

Essay Using DNA in science and technology

12

DNA and classification

2.2 Structure of DNA

2.3 Differences in DNA lead to genetic diversity

2.9 Comparison of DNA base sequences

Genetic engineering and making useful substances

2.5 Plasmids

5.8 The use of recombinant DNA to produce transformed organisms that benefit humans

Other uses of DNA

2.5 Cell cycle and treatment of cancer

5.8 Gene therapy;

Medical diagnosis and the treatment of human disease;

The use of DNA probes to screen patients for clinically important genes.

(a) (i) Spindle formed / chromosome / centromere / chromatids

13

attaches to spindle;

Chromosomes / chromatids line up / move to middle / equator (of cell);

Do not award second mark for answers referring to chromosomes 'pairing up'.

Ignore reference to homologous chromosomes unless context suggests pairing which negates second mark.

Neutral: Details on nuclear membrane.

Accept: Diagram for second marking point.

2

(ii) Chromosome / centromere splits / chromatids / 'chromosomes' separate / pulled apart;

To (opposite) sides / poles / centrioles (of cell);

Reject: Homologous chromosomes separate for first marking point.

Accept: Diagram for second marking point.

Chromatids / 'chromosomes' move to poles / sides / centrioles = 2 marks.

2

(b) (i) Form / replace cells quickly / rapidly / divide / multiply / replicate rapidly;*Neutral: Repair cells.*

Answers must convey idea of 'speed'.

1

(Ii) Correct answer = 774 minutes / 12 hours 54mins = 2 marks;;

Incorrect answer but indicates 3 cell cycles involved = one mark;

2

(c) Prevents / slows DNA replication / doubling / prevents / slows mitosis;

New strand not formed / nucleotides (of new strand) not joined together / sugar-phosphate bonds not formed;

First marking point must be in context of DNA replication not cell replication.

Do not negate first marking point if role of DNA polymerase is described incorrectly e.g. Reject: 'joins bases / strands together'. Role of DNA polymerase must be correct for last marking point.

2

[9]

14 (a) In one country where the percentage of fat (in the diet) is 35%, the death rate (from breast cancer) is 20 per 100 000;

Must have reference to country

Accept … 1 per 5 000 / 0.02%

1

(b) 1. No. of deaths from breast cancer divided by total population × 100 000;

2. No. of deaths from breast cancer divided by all deaths × 100 000;

3. Sample and count deaths from breast cancer in 100 000 people;

If sample not 100 000 then must scale appropriately

1 max

(c) 1. Positive correlation;

2. But correlation does not show causation / some other (named) factor may beinvolved;

3. Evidence against positive correlation e.g. different death rates at same % fat /similar death rates at different % fat / some countries with higher death rate have lower fat intake;

1. Accept description of positive correlation / directly proportional.

Accept positive relationship.

2. Do not accept casual in place of causal.

3. Answer must be consistent with data.

3

[5]

15

(a) (i) Increases then plateaus / constant / steady / rate does not change;

Neutral: 'peaks' / 'reaches a maximum' / 'stops increasing' / 'no effect' instead of 'plateaus'
Reject: rate decreases / reaction stops

Correct reference. to 27 / 28 units;
e.g. increases up to / plateaus at 27 / 28

2

(ii) Substrate concentration / amount of substrate;

As substrate concentration increases, rate increases / positive correlation (between rate and substrate concentration);

2

(iii) All <u>active sites</u> occupied / saturated / enzyme limiting (rate of reaction) / maximum number of E-S complexes;

Reject: enzymes used up
Reject: substrate limits rate of reaction
Neutral: substrate no longer limits the reaction
Neutral: reference to temperature

1

(b) Curve is lower and plateaus at a higher substrate concentration (it must also start at zero);

Accept: curve lower and joins existing curve at final point (with no plateau)
Reject: if curve plateaus before original
Reject: if curve plateaus lower than original

1

(c) (i) Methotrexate / drug is a similar shape / structure to substrate so binds to / fits /is complementary to active site;

Q *Reject: same structure / shape*

Q *Reject: reacts with active site*

Less substrate binds / less enzyme-substrate complexes formed;

Accept: substrate cannot bind / enzyme-substrate complex not formed

2

(ii) Methotrexate / drug is only similar shape to specific substrate / only fits this <u>active site</u>;

Assume that 'it' refers to the drug

OR

Methotrexate / drug is a different shape to other substrates / will not fit other <u>active sites</u>;

1

[9]

16

(a) To ensure the colour is the same at the start;

1

(b) Yes – curve on graph with bromelain present remains approximately constant / risesvery slightly;
Would decrease if killing of cells occurred / would increase if cells still dividing;

2

(c) Use of mouse cells (rather than human);
(Carried out) *in vitro* / not in living organisms;
Only tested on one type of cancer;
Not possible to predict effect on humans (as no data collected);

3 max

(d) The faster the rate of division the faster the cancer would grow;
By measuring rate of cell division you could see how effective the treatment was;

2

(c) Not ethical to replace conventional treatment;
As life of patient is at risk (if bromelain not effective);

2

[10] (a)

17

Nucleus	Number of chromosomes	Mass of DNA / arbitrary units
At telophase of mitosis	26;	30;
From a sperm cell	13;	15;

4

(b) Cancer cells often have faulty / damaged DNA;

Protein / p53 faulty / not made;

Cell (with faulty / DNA) divides / completes cell cycle;

Uncontrolled division produces cancer;

p53 refers to the protein so do not accept reference to p53 mutating.

3

(c) (i) Interphase / S phase / synthesis phase;

1

(ii) Anaphase / **A**;

1

[9]

(a) 1 Cut gene out of cell / make gene using mRNA / obtain gene with restriction enzymes;

18

2 Cut DNA using restriction enzyme / plasmid cut with restriction enzyme;

3 Correct reference to sticky ends;

4 Join DNA using ligase / insert gene into vector;

5 Plasmid / named vector transferred to cell;

6 Method of transfer e.g. heat shock;

7 Reference to marker gene;

8 Select bacteria containing new gene;

max 6

(b) Cells can metastasise / break off / spread to other parts of the body;

Remaining cells continue to divide forming a new tumour / secondary;

2

(c) Antibodies specific;

Normal cells have different antigen / cancer cell has particular antigen;

Enzyme **only** present in cancer cells so drug **only** activated at / near cancer cells;

3

(d) All cells contain DNA;
Would stop / inhibit DNA replication in normal cells;
Stops / inhibits cell division;
Named example on growth / repair e.g. no new blood cells made / no wound healing;

4

[15]

19 (a) (i) benign does not cause cancer / does not invade other tissues causing damage / with benign cancer, pieces which break off do not start new tumours elsewhere in body / metastasis;

1

(ii) may damage organ concerned; may cause blockages / obstructions; may damage / exert pressure on other organs;

max 2

(b) (i) because sun's radiation contains ultra violet radiation; this causes mutation of genes which control division;

2

(ii) because fair skin has little melanin which protectsagainst u.v. radiation;

1

(iii) because cancer has genetic component / may have inherited

(onco)gene / gene which gives predisposition to / causes cancer;

1

[7] (a) 1 (DNA altered by) mutation;

20

2 (mutation) changes base sequence;
3 of gene controlling cell growth / oncogene / that monitors cell division;
4 of tumour suppressor gene;
5 change protein structure / non-functional protein / protein not formed;
6 (tumour suppressor genes) produce proteins that inhibit cell division;7 mitosis;
8 uncontrolled / rapid / abnormal (cell division);
9 malignant tumour;

max 6

(b) cancer cells die / break open;releasing DNA;

2

(c) normal DNA and changed DNA have different sequences;
DNA only binds to complementary sequence;

2

(d) fewer abnormal / cancerous cells / smaller tumours;less cell damage / less spread / fewer locations to treat;

2

(e) mRNA base sequence has changed;gene / DNA structure is different / has mutated; cancer gene active / tumour suppressor gene inactive;

3

[15] (i) Because there are big differences;

21

any correct named example e.g. lung cancer / bronchitis much lower in women than in men;

2

(ii) easier to compare if sample size effectively the same;
different numbers of people in each group;

2

[4]

(a) secreted by the liver / storage / release from gall bladder into the duodenum / small

22

intestine;
bile passes unchanged from small intestine to colon;

2

(b) (i) chance alone has not caused the difference (between the two patients types);high steroid high bacteria (significantly) higher percentage of cancer patients / low steroids low bacteria (significantly) higher percentage of control patients;

2

(ii) some patients with low levels of one / both factor(s) have cancer;

1

(c) change in code / base sequence / structure of gene;addition / deletion / substitution; mRNA / transcription changed; gene product / protein structure / amino acid sequence changed / different protein; loss of function; uncontrolled cell division;

4 max

[9] (a) mass of undifferentiated / unspecialised / totipotent cells;

23

uncontrolled cell division;
(not 'repeated')

metastasis / (cells break off and) form new tumours / spread to other parts of body;

3

(b) cancer takes time to develop / exposure when young but cancertriggered later; other organs destroyed before death occurs / metastasis affects other organs; immune system less effective in old people;
longer time of exposure to UV / accumulation of mutagenic effect;

1 max

(c) dark skin / melanin / pigment stops UV light / prevents burning;so less cancer risk in dark skinned people / less likely to develop tumours; *(allow converse)*

2

[6] (i) smoking and drinking increase risk;

24

risk increases for nonsmokers with more alcohol;
20-40 cigarettes increases risk; at all levels of alcohol consumption; 4 or more drinks increase risk in all groups; worst

risk with combination of 40+ cigarettes and 4 or more drinks;
smoking and drinking together have a greater effect than either on its own; over 40 cigarettes and no alcohol greater than 1 or 2 alcoholic drinks / valid comment about anomaly;

3 max

(ii) other environmental factor / e.g. passive smoking;
genetic predisposition / inherited from parents;
mutation;

1 max

[4] (a) Daughter (C) does not have the condition / one child doesn't have it;

25

Accept converse arguments (If candidates see it purely as a genetic cross diagram) D is heterozygous because E is unaffected;

Parents must have been carriers of normal / healthy recessive/ if recessive then parents homozygous (so all children affected);

D has cancer, so the cancer allele must be dominant;

2

(b) Father (A) would pass on X chromosome to daughter;
She is not affected;

Accept that if D's X chromosome carried 'it', then E would be affected.

2

(c) Only 25 / young so don't know if cancer will develop;

Accept E must be homozygous recessive/have two recessive alleles;

Don't know if her father was heterozygous or homozygous;

So no chance of cancer / no more chance than rest of the population;

If heterozygous, she has a 50% chance of carrying the allele/gene;
If homozygous, she has a serious risk of cancer.

2 max

(d) Mutation / mutagen changes DNA of cell;
Damaged DNA not repaired / cells not killed / apoptosis doesn't happen; Mutation leads to loss of control / uncontrolled cell division;
(Some of these) cells carried to other parts of the body.

3 max

[9] (a) No cadmium;

26

Other conditions same as cadmium-treated group;

2

(b) (i) As a measure of the effect due to cadmium / to make a comparison;

1

(ii) Becoming more methylated;

Ignore later slight decrease/no change

1

(iii) Production of more methyltransferase enzyme /increased activity of transferase;

Extra incorrect relevant information - cancel

1

(c) RNA-polymerase could not bind (to DNA / to promoter);mRNA of p16 could not be made / no transcription of p16 gene;

2

(d) Any **four** from:

1. Cadmium causes expression of methyltransferase gene / increased activity transferase (from 2 to 3 weeks in);
2. Methyl groups on to promoter / p16 gene / suppressor (gene);
3. (p16) normally suppresses tumour growth;
4. p16 protein / p16 expression falls after 4 weeks / after methylation; 5. Tumour formation occurs (after 10 weeks) after p16 falls / after suppressor gene activity falls;

4 max

[11]