Q1.

(a) In the UK in 2016, there were 525 048 deaths. Cancer caused 30.4% of all deaths. Throat cancer caused 5% of all deaths from cancer.

Calculate the mean number of people who died of throat cancer per month in 2016.

Show your working.

Answer _____ people per month

(2)

Increased methylation of the promoter region of a tumour suppressor gene causes one type of human throat cancer.

In this type of throat cancer, cancer cells are able to pass on the increased methylation to daughter cells. The methylation is caused by an enzyme called DNMT.

Scientists have found that a chemical in green tea, called EGCG, is a competitive inhibitor of DNMT. EGCG enables daughter cells to produce messenger RNA (mRNA) from the tumour suppressor gene.

(b) Suggest how EGCG allows the production of mRNA in daughter cells.

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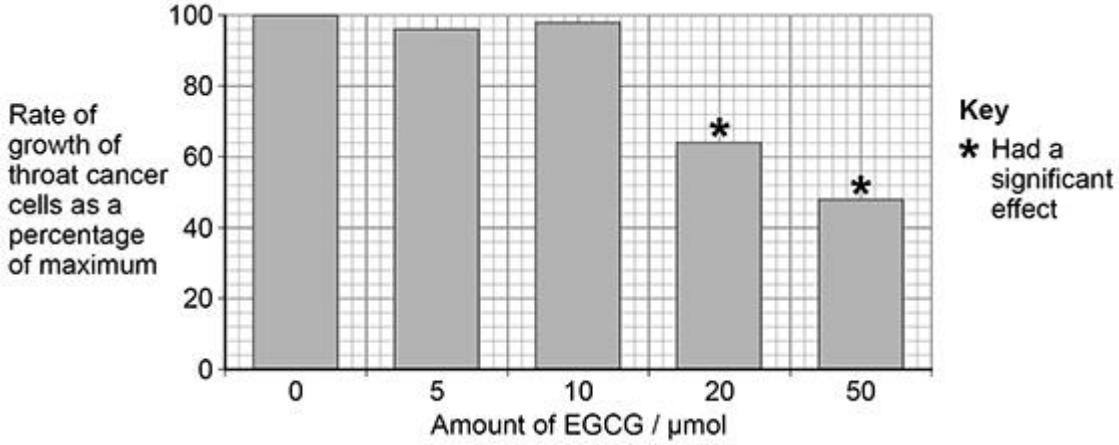
(3)

The scientists investigated the effect of different amounts of EGCG on the growth rate of the throat cancer cells grown *in vitro*. Their results are shown in the graph below.



(c) A reporter who reviewed all of this work concluded that drinking green tea could be a cure for cancer.

Suggest **three** reasons why his conclusion might **not** be valid.

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(3)

(Total 8 marks)

Q2.

(a) There are different types of gene mutation.

Put a tick (✓) in the box next to the statement which describes **incorrectly** the effect of the mutation in an exon of a gene.

A substitution may not result in a change to the encoded amino acid.	☐
An inversion will result in a change in the number of DNA bases.	☐
A deletion will result in a frame shift.	☐
An addition will result in a frame shift.	☐

(1)

(b) Describe how alterations to tumour suppressor genes can lead to the development of tumours.

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(3)

(c) A type of malignant tumour cell divides every 8 hours.

Starting with one of these cells, how many tumour cells will be present after 4 weeks?
Assume none of these cells will die.

Give your answer in standard form.

Answer = _____

(2)

(Total 6 marks)

Q3.

(d) Sometimes, a mutagenic agent causes DNA to break. A different enzyme called ATM binds to the broken DNA. This leads to the activation of a protein coded for by a tumour suppressor gene. The effect of ATM binding is to stop cell division until DNA is repaired.

A mutation could result in a person having non-functional forms of the gene that produces ATM.

What can you predict about the possible effects of having a non-functional form of ATM?

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(3)

Q4.

(a) Define what is meant by epigenetics.

(2)

(b) In eukaryotes, transcription of target genes can be stimulated or inhibited when specific transcriptional factors move from the cytoplasm into the nucleus.

Oestrogen, methyl groups and acetyl groups are control factors that can play a role in initiating transcription.

Complete the table to show features of these control factors.

Put a tick (✓) in the box if the control factor shows the feature.

	Feature	
Control factor	**Binds with DNA**	**Binds with protein**
Oestrogen		
Methyl groups		
Acetyl groups		

(2)

(c) Explain how increased methylation could lead to cancer.

(3)

(d) Give **one** way in which benign tumours differ from malignant tumours.

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(1)

(Total 8 marks)

Q5.

(a) Explain how the methylation of tumour suppressor genes can lead to cancer.

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(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 of graph.

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(3)

(c) What can you conclude from these data?

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(2)

(Total 8 marks)

Q6.

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?

(2)

(b) In many trials of new drugs, a control group of patients is given a placebo that does not contain 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 treatment of MM.

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

(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.

(3)

(Total 10 marks)