**Q1.**

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

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**(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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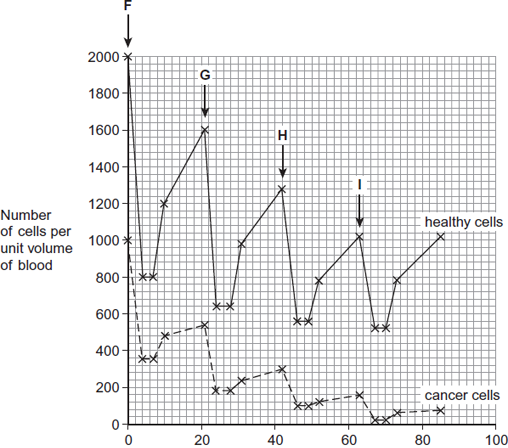
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**(3)**

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.

                                    Time / days



(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 the drug between times **F** and **G**.

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**(3)**

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

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

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**(2)**

**(Total 15 marks)**

**Q2.**

Phenylketonuria is a disease caused by mutations of the gene coding for the enzyme PAH. The table shows part of the DNA base sequence coding for PAH. It also shows a mutation of this sequence which leads to the production of non-functioning PAH.

|  |  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| DNA base sequence coding for PAH | C | A | G | T | T | C | G | C | T | A | C | G |
| DNA base sequence coding for non-functioning PAH | C | A | G | T | T | C | C | C | T | A | C | G |

(a)     (i)      What is the maximum number of amino acids for which this base sequence could code?



**(1)**

(ii)     Explain how this mutation leads to the formation of non-functioning PAH.

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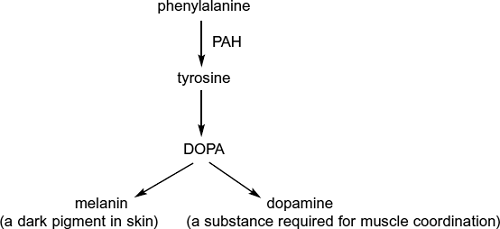
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**(3)**

PAH catalyses a reaction at the start of two enzyme-controlled pathways.  
The diagram shows these pathways.



(b)     Use the information in the diagram to give **two** symptoms you might expect to be visible in a person who produces non-functioning PAH.

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**(2)**

(c)     One mutation causing phenylketonuria was originally only found in one population in central Asia. It is now found in many different populations across Asia. Suggest how the spread of this mutation may have occurred.

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**(1)**

**(Total 7 marks)**

**Q3.**

Testosterone is a steroid hormone that belongs to a group of male sex hormones called androgens.

(a)     Steroid hormones are hydrophobic.

Explain why steroid hormones can rapidly enter a cell by passing through its cell-surface membrane.

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**(2)**

(b)     In the cytoplasm, testosterone binds to a specific androgen receptor (AR).

An AR is a protein.

Suggest and explain why testosterone binds to a specific AR.

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**(2)**

(c)     The binding of testosterone to an AR changes the shape of the AR. This AR molecule now enters the nucleus and stimulates gene expression.

Suggest how the AR could stimulate gene expression.

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**(2)**

The gene that codes for the AR has a variable number of CAG repeats.

Some studies have shown an association between the number of CAG repeats and the risk of developing prostate cancer.

The table below shows the results of a statistical test from one study.

|  |  |
| --- | --- |
| **Number of CAG repeats in the** ***AR*** **gene** | **Probability (P) value** |
| ⩽ 16 | 0.02 |
| ⩽ 17 | 0.30 |
| ⩽ 18 | 0.07 |
| ⩽ 19 | 0.09 |
| ⩾ 20 | 0.06 |

(d)     What can you conclude from the data in the table above?

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**(3)**

**(Total 9 marks)**

**Q4.**

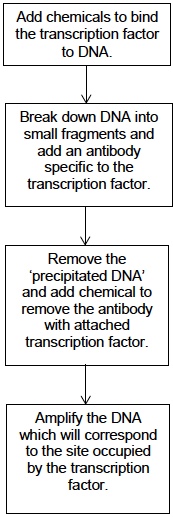
(a)     What is meant by a genome?

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**(1)**

Chromatin immunoprecipitation is one method to determine where a transcription factor binds to DNA. The principle behind this procedure is shown in the chart.



(b)     Explain why the antibody binds to the transcription factor.

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**(2)**

(c)     Use the chart to explain what ‘precipitated DNA’ consists of.

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**(1)**

Soybeans are used in a number of processed foods. However, soybeans contain a protein known as P34 that causes an allergic response in some people. Scientists have created transgenic soybeans that produce single-stranded cDNA, which prevents transcription of the *P34* gene. They used recombinant plasmids as vectors to transform soybean cells. After they had screened these cells for production of the P34 protein, they cultured the transformed cells to form soybean plants.

(d)     Suggest how single-stranded cDNA could prevent transcription of the *P34* gene.

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**(1)**

(e)     Describe the roles of **two** named types of enzymes used to insert DNA fragments into plasmids.

Type of enzyme \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

Role \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

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Type of enzyme \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

Role \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

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**(2)**

(f)      The soybean cells were screened for the presence of the P34 protein. This process involved the use of gel electrophoresis to separate proteins extracted from soybean cells.

Suggest **two** features of the structure of different proteins that enable them to be separated by gel electrophoresis.

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**(2)**

**(Total 9 marks)**

**Q5.**

(a)     Each year, a few people with type I diabetes are given a pancreas transplant. Pancreas transplants are not used to treat people with type II diabetes.

Give **two** reasons why pancreas transplants are not used for the treatment of type II diabetes.

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**(2)**

(b)     The pancreas produces the hormone insulin.

Put a tick (✔) in the box next to the statement which describes **incorrectly** the action of insulin.

|  |  |
| --- | --- |
| Activates enzymes involved in the conversion of glucose to glycogen. |  |
| Controls the uptake of glucose by regulating the inclusion of channel proteins in the surface membranes of target cells. |  |
| Attaches to receptors on the surfaces of target cells. |  |
| Activates enzymes involved in the conversion of glycerol to glucose. |  |

**(1)**

(c)     Scientists investigated the use of induced pluripotent stem cells (iPS cells) to treat type I diabetes in mice. The scientists used four transcription factors to reprogramme skin cells to form iPS cells. The scientists then stimulated the *in vitro* differentiation of iPS cells into pancreatic cells.

The scientists set up three experimental groups:

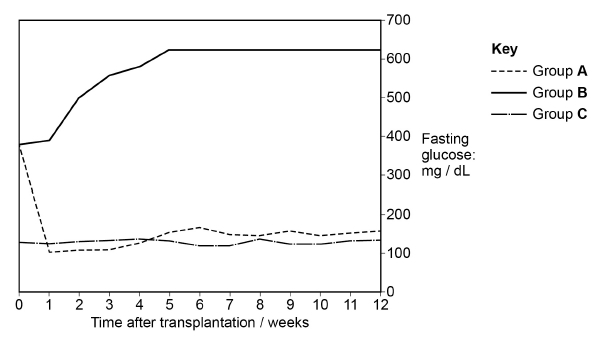
•   Group **A** – 30 mice with type I diabetes received pancreatic cell transplants derived from iPS cells.

•   Group **B** – 30 mice with type I diabetes were left untreated.

•   Group **C** – 30 mice without diabetes were left untreated.

The scientists measured the blood glucose concentration of all the mice on a weekly basis for 12 weeks.

The results the scientists obtained are shown in the graph.



Suggest how transcription factors can **reprogramme** cells to form iPS cells.

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**(2)**

(d)     Using all the information provided, evaluate the use of iPS cells to treat type I diabetes in humans.

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**(4)**

**(Total 9 marks)**

**Q6.**

Alzheimer’s disease (AD) is a non-reversible brain disorder that develops over a

number of years. At the start of 2014 the number of Americans with AD was

estimated to be 5.4 million. Every 30 seconds another person in America

develops AD.

5        In the brain of a person with AD there is a lower concentration of acetylcholine.

This affects communication between nerve cells and initially results in memory

loss and confusion. Some of the symptoms of AD that are associated with

communication between nerve cells are reduced by taking the drug donepezil.

Donepezil inhibits the enzyme acetylcholinesterase.

10      A gene mutation called E280A found on chromosome 14 causes early-onset AD

at a mean age of 49 years. The age at which the E280A mutation is expressed

to cause AD varies.

Yaramul is a town in a historically isolated region of the Andes Mountains. The

population of this town has the highest frequency of the E280A mutation in the

15      world. The origin of the E280A mutation in this population has been traced back

to a common ancestor in the 17th century. Natural selection has not reduced

the frequency of the E280A mutation in the population.

This autosomal dominant mutation involves a change in triplet 280 from GAA to

GCA. Scientists analysed chromosome 14 from 102 individuals from Yaramul.

20      They recorded a sample size of 204 and detected 75 E280A mutations but only

74 potential AD cases. The scientists identified individuals with the mutation by

whole genome sequencing. They had decided that a DNA probe would not be a

suitable method to detect the E280A mutation.

(a)     Assuming no one with AD died in 2014, calculate the annual percentage increase in AD cases in America for 2014 (lines 2–4).

Answer = \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ %

**(2)**

(b)     Explain how donepezil could improve communication between nerve cells (lines 7–9).

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**(3)**

(c)     Suggest and explain **two** reasons why there is a high frequency of the E280A mutation in Yaramul (lines 13–15).

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**(2)**

(d)     Explain why natural selection has **not** reduced the frequency of the E280A mutation in the population (lines 16–17).

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**(2)**

(e)     The age at which the E280A mutation is expressed to cause AD can vary (lines 11–12).

Suggest and explain **one** reason for this.

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**(2)**

(f)      One scientific study which analysed chromosome 14 involved 102 individuals. The scientists recorded a sample size of 204. In this sample they detected 75 E280A mutations but only 74 potential AD cases (lines 19–21).

Suggest explanations for the figures the scientists recorded.

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**(2)**

(g)     Suggest why a DNA probe for the mutated triplet was **not** considered a suitable method for detection of the E280A mutation (lines 22–23).

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**(2)**

**(Total 15 marks)**

**Q7.**

Scientists have investigated the use of different types of stem cell to treat damage to the heart after a myocardial infarction. During a myocardial infarction, a number of different cell types in the heart die. This includes cardiomyocytes which are heart-muscle cells.

Embryonic pluripotent stem cells (ESCs) can divide and differentiate into a wide range of different cell types.

(a)     Using the information given, suggest **one** reason why ESCs might be suitable to treat damage to the heart.

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**(1)**

(b)     ESCs have not yet been used to treat people who have had a myocardial infarction. This is because of concern that the use of ESCs might lead to more harm to the person. One way that ESCs might lead to more harm is by differentiating into the wrong types of cells.

Suggest **one** other way that putting ESCs into a person’s heart might lead to more harm to the person.

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**(2)**

(c)     Transplants of cardiomyocytes have been shown to increase the repair of heart tissue damaged by myocardial infarction.

One group of scientists investigated the hypothesis that these transplants work by stimulating growth of new blood vessels into damaged heart tissues. They obtained three groups of mice, **A**, **B** and **C** that had suffered myocardial infarctions.

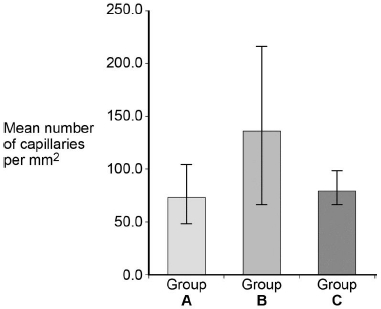
•        **Group A** were operated on but no transplant was given.

•        **Group B** were operated on and given transplants containing cardiomyocytes and two other types of heart cell.

•        **Group C** were operated on and given transplants containing the two other types of heart cells but no cardiomyocytes.

After a suitable time, the scientists measured the mean number of capillaries per mm2 in sections taken from areas of the hearts of the mice affected by myocardial infarction.

Their results are shown in the graph below. The bars show ±2 standard deviations, which includes 95.4% of the data.



Group **A** was a control group. Explain **two** ways in which Group **A** acts as a control.

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**(2)**

(d)     What can you conclude from these data about the stimulation by cardiomyocytes on growth of new blood vessels into damaged heart tissues?

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**(3)**

(e)     Suggest how the growth of new blood vessels into damaged heart tissues could increase the rate of repair of tissues.

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**(3)**

(f)      The scientists used an optical microscope to measure the number of capillaries in thin sections cut from samples of heart muscle.

Describe the method they would have used to find the mean number of capillaries per mm2.

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**(4)**

**(Total 15 marks)**

**Q8.**

(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)**

**Q9.**

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 not contain any drug.

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

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**(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.

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**(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.

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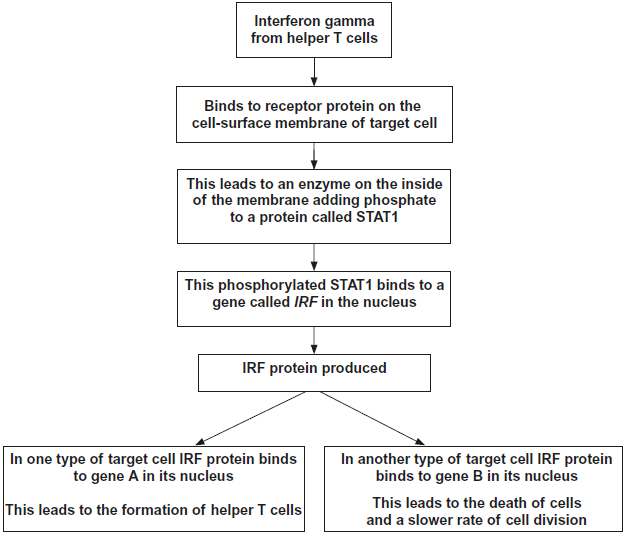
**(3)**

**(Total 10 marks)**

**Q10.**

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 its receptor protein leads to the production of phosphorylated STAT1.

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**(2)**

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

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2. \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

**(2)**

(c)     The regulation of the formation of helper T cells by interferon gamma is an example of positive 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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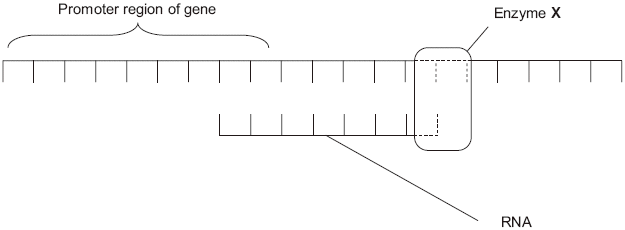
**(3)**

**(Total 9 marks)**

**Q11.**

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

**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 receptor in 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.

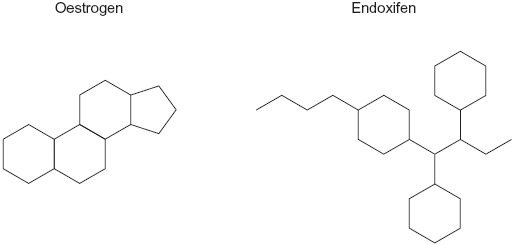
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**(1)**

(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**



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

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**(2)**

**(Total 6 marks)**

**Q12.**

Plant physiologists attempted to produce papaya plants using tissue culture. They investigated the effects of different concentrations of two plant growth factors on small pieces of the stem tip from a papaya plant. Their results are shown in the table.

|  |  |  |  |
| --- | --- | --- | --- |
| **Concentration of auxin / μmol dm–3** | **Concentration of cytokinin / μmol dm–3** | | |
| 5 | 25 | 50 |
| 0 | No effect | No effect | Leaves produced |
| 1 | No effect | Leaves produced | Leaves produced |
| 5 | No effect | Leaves produced | Leaves and some plantlets produced |
| 10 | Callus produced | Leaves and some plantlets produced | Plantlets produced |
| 15 | Callus produced | Callus and some leaves produced | Callus and some leaves produced |

Callus is a mass of undifferentiated plant cells. Plantlets are small plants.

(a)     Explain the evidence from the table that cells from the stem tip are totipotent.

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**(2)**

(b)     Calculate the ratio of cytokinin : auxin that you would recommend to grow papaya plants by this method.

Answer \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

**(2)**

(c)     (i)      Papaya plants reproduce sexually by means of seeds. Papaya plants grown from seeds are very variable in their yield. Explain why.

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**(2)**

(ii)     Explain the advantage of growing papaya plants from tissue culture rather than from seeds.

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**(1)**

**(Total 7 marks)**

Mark schemes

**Q1.**

(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 for amino 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 percentage decrease in number of cancer cells / greater proportion of cancer cells killed*

4.      Greater / faster increase in number of healthy cells / more healthy cells replaced / 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]**

**Q2.**

(a)     (i)      4;

**1**

(ii)     1.      Change in amino acid / (sequence of) amino acids / primary structure;

*1. Reject = different amino acids are 'formed'*

2.      Change in hydrogen / ionic / disulphide bonds alters tertiary structure / active site (of enzyme);

*2. Alters 3D structure on its own is not enough for this marking point.*

3.      Substrate not complementary / cannot bind (to enzyme / active site) / no enzyme- substrate complexes form;

**3**

(b)     1.      Lack of skin pigment / pale / light skin / albino;

2.      Lack of coordination / muscles action affected;

**2 max**

(c)     Founder effect / colonies split off / migration / interbreeding;

*Allow description of interbreeding e.g. reproduction between individuals from different populations*

**1**

**[7]**

**Q3.**

(a)  1.      Lipid soluble;

*Ignore ‘not water soluble’ or ‘fat soluble’.*

2.      (Diffuse through) phospholipid (bilayer);

*Ignore reference to joining to receptors/channels/carriers but reject passage through protein channels/carriers.*

**2**

(b)     1.      Has a (specific) tertiary structure/shape;

*Accept in context of AR or testosterone.*

*Ignore 3D.*

2.      (Structures are) complementary;

*Reject reference to antigen.*

*Reject reference to active site, enzyme, substrate or induced fit.*

**2**

(c)     1.      (AR is) a transcription factor;

*Ignore ‘binds to bases’ or ‘binds to gene’.*

2.      Binds to DNA/promoter;

*Reject reference to active site, enzyme, substrate or induced fit.*

3.      (Stimulates) RNA polymerase;

**2 max**

(d)     1.      With 16 or fewer than 16 (repeats the association) is significant;

*If none of the marks is awarded allow principle mark of (prostate) cancer more likely with 16 or less than 16 (repeats)* ***or*** *(prostate) cancer less likely with 17 or more than 17 (repeats)*

***OR***

*Alternative principle mark Correctly links significant/not significant to correct probability value/percentage* ***or*** *to rejecting/accepting the null hypothesis.*

*Reject ‘the results are significant’.*

*Accept ‘difference in results is significant’.*

2.      \*With 17 or more than 17 (repeats the association) is **not** significant;

3.      \*With 16 or fewer than 16 (repeats) there is less than a 5% or less than 0.05 probability of being due to chance

**OR**

\*With 17 or more than 17 (repeats) there is more than a 5% or more than 0.05 probability of being due to chance

**OR**

\*Explanation of a probability value e.g. 0.30 is a 0.30 or 30% probability of being due to chance;

*Accept equivalent responses in terms of 95% or 0.95 probability.*

4.      \*With 16 or fewer than 16 (repeats) reject the null hypothesis

**OR**

\*With 17 or more (repeats) accept the null hypothesis;

*\*Accept reference to any number of repeats (e.g. 18) between 17 to 20 for 17 or more than 17 (repeats).*

**3 max**

**[9]**

**Q4.**

(a)     (All) the DNA in a cell/organism;

*Accept*

*‘(all) the ‘genes’/alleles’ ‘genetic material/code’ in a cell/organism/ person’*

*‘the total number of DNA bases in a cell/organism’*

*Reject all the DNA/ genes within a species*

**1**

(b)     1.      (Transcriptional factor/antibody) has a specific/tertiary structure/shape;

*Accept (antibody) has a specific variable region*

*Accept (transcription factor/antibody) has a specific binding site*

*Reject active site but only once.*

2.      Complementary (shape/structure);

*Reject active site but only once.*

**2**

(c)     DNA, transcription factor and antibody;

*Accept Nucleotides for DNA*

*Ignore ‘reference to chemicals’*

**1**

(d)     Binds to P34 gene/DNA/mRNA

**OR**

Binds to transcription factor gene/DNA

**OR**

Binds to promoter;

*Reject binds to transcription factor*

**1**

(e)     1.      Restriction (endonuclease/enzyme) to cut plasmid/vector;

2.      Ligase joins gene/DNA to plasmid/vector;

**2**

(f)     1.      Mass/number of amino acids/polypeptides;

*Accept weight for mass*

*Ignore density/size*

*Accept length of polypeptide/amino acid chain*

*Accept primary structure /sequence of amino acids.*

*Accept tertiary structure*

2.      Charge;

3.      R groups (differ);

**2 max**

**[9]**

**Q5.**

(a)     1.      (Usually)Type II produce insulin;

2.      Cells / receptors less sensitive / responsive (to insulin)

**OR**

Faulty (insulin) receptors;

3.      (Treated / controlled by) diet / exercise;

*2.      Accept: cells / receptors do not respond.*

*2.      Accept: ‘fewer receptors’*

*3.      Accept: (Treated / controlled by) weight loss / medication / drugs.*

*3.      Ignore: diabetes is caused by diet / exercise.*

**2 max**

(b)     Tick in box 4

**1**

(c)     1.      Attach to gene / DNA / promoter region;

2.      Stimulate / inhibit transcription / RNA polymerase;

*Note: Genes being expressed / inhibited or switched on / off is not enough on its own.*

**2**

(d)     1.      (Effective as) group A / with iPS / treated lower than group B / with diabetes;

2.      (Effective as) group A similar to group C / without diabetes;

3.      (Investigation) done on mice not humans;

4.      Only shows results for 12 weeks / short-time period / long-term effects not known;

*Ignore: Only one study / not repeated / sample size.*

*2.      Accept: ‘healthy’ or ‘normal’ or control for group C.*

**4**

**[9]**

**Q6.**

(a)     1.      Correct answer of 19.4 / 19.41%

**OR**

19.47 / 19.5% = **2 marks**;

2.      Incorrect answer but shows increase of

1,048,320 **OR** 1,051,200 = one mark;

*Accept: 19.46% for one mark.*

**2**

(b)     1.      Less / no acetylcholine broken down;

2.      Acetylcholine attaches to receptors;

3.      (More) Na+ enter to reach threshold / for depolarisation / action potential / impulse;

*1.      Accept: more acetylcholine present / remains.*

*1 and 2. Accept: remains attached for longer = 2 marks.*

*3.      Must be sodium ions.*

**3**

(c)     1.      Isolated **so** inbreeding / low genetic diversity / small gene pool;

2.      Allele inherited (through generations) from (common) ancestor;

*1.      Ignore: Founder effect.*

*1.      Accept: no interbreeding with other populations.*

*1.      Reject: interbreeding within the population.*

**2**

(d)     1.      AD / symptoms develops late / at 49;

2.      Have already reproduced;

*Note: ‘It’ is not equivalent to AD / symptom as the question stem relates to the mutation.*

**2**

(e)     1.      Epigenetics / environment / named factor e.g. stress, alcohol, toxins, diet, exercise, smoking;

2.      methylation (of genes)

**OR**

acetylation (of histones);

*1.      Ignore: gender and lifestyle.*

*2.      If further details are provided the context must be correct e.g. increased methylation or decreased acetylation inhibit gene expression / transcription.*

**2**

(f)      1.      One person was homozygous dominant / has two dominant alleles = **2 marks**;

2.      For one mark has two alleles / chromosomes;

*1.      Accept; homozygous dominant genotype e.g. ‘one person has AA’ for 2 marks.*

*2.      Accept: is diploid or has two copies of the gene.*

**2**

(g)     1.      (GCA / triplet) is common / found in other places;

2.      Would not know if it was the mutation / allele / gene

**OR**

Produces ‘false positives’

*1.      Accept: Probe will bind elsewhere.*

**2**

**[15]**

**Q7.**

(a)     1.      (ESCs) can replace any type of (heart) cell;

*Accept named type of cell, e.g. heart muscle cell*

**1**

(b)     1.      Might divide out of control;

2.      Leading to tumour / cancer;

**2**

(c)     1.      Shows the effects of surgery;

2.      Allows effects of transplants / treatment to be seen;

*Allow in either order*

**2**

(d)     1.      Other cell types cause some increase but most of increase due to cardiomyocytes;

2.      Large SD, so some not much increase / no better than control;

3.      Overlap of SDs indicates / suggests no significant difference;

**3**

(e)     1.      Greater blood supply (to damaged areas);

2.      Bringing more oxygen / glucose for respiration;

3.      Brings more amino acids for protein synthesis;

4.      For cell repair / mitosis / division;

**3 max**

(f)     1.      Measure diameter of field of view and calculate area;

2.      Using micrometer slide and eyepiece graticule;

*Accept descriptions*

3.      Count number of capillaries in large number of fields of view and calculate mean;

4.      Select fields of view randomly

**4**

**[15]**

**Q8.**

(a)     1.      Methylation prevents transcription of gene;

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 death rate from breast cancer;

2.      But number of points off line / anomalies.

**2**

**[8]**

**Q9.**

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

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% to 15.2%;

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 some patients;

6.      Quality of (extra) time alive not reported;

*If answers relate only to ‘Yes’ or ߢNo’, 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]**

**Q10.**

(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/process*

*3. 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 of damaged/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 OR slows 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]**

**Q11.**

(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 RNA polymerase / 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]**

**Q12.**

(a)     1.      Gives rise to new plants / plantlets;

2.      So must be able to develop into different tissues / other specialised cell types / differentiate;

*1. Ignore references to leaves / callus*

**2**

(b)     Two marks for 5 : 1/50 : 10/1 : 0.2;;

*One mark for ratio correctly identified but expressed incorrectly as 1 : 5 / 10 : 50 / 0.2 : 1;*

**2**

(c)     (i)      1.      Meiosis / independent assortment / crossing over;

2.      (Fusion of) genetically different gametes / random fertilisation;

**2**

(ii)     Will be clones / produced by mitosis / will be genetically identical / less variation / all plants will have desired characteristics;

*If the reference is to identical must be genetically identical, but allow less variation without the reference to genetical.*

**1**

**[7]**

Examiner reports

**Q1.**

Parts (a), (b) and (d) proved to be good discriminators.

(a)     It was disappointing that only just below 40% of students scored at least half marks. This was mainly due to simply describing the structure of DNA, without explaining how these features relate to its functions. Some students wrote about DNA structure and function in different paragraphs. This made it unclear which feature went with which function, as no direct links had been made. In contrast, there were some truly excellent responses, which had clearly been well planned before putting pen to paper. The most common mark points awarded were for the sugar-phosphate backbone providing strength or protecting bases, the helix allowing the molecule to be compact, weak hydrogen bonds allowing strand separation or replication and the two strands acting as templates or allowing semi-conservative replication. Relatively few students linked complementary base pairing with accurate replication or the production of identical copies of DNA. Similarly, few students referred to DNA as a large molecule that can store lots of information, or the base sequence coding for amino acids. Weaker responses often mentioned this in the context of the genetic code being degenerate. Indeed, some students thought that the base sequence causes amino acids to be *produced*. The ability to convey that *many* hydrogen bonds provide stability was rarely seen. It was also unfortunate that a number of students wasted their time by writing about irrelevant topics such as the differences between prokaryotic and eukaryotic DNA and the role of histones. There were also some lengthy accounts of DNA replication, enzyme structure and the different levels of protein structure.

(b)     Many students scored at least two marks for stating that a mutation in gene **E** produces the highest risk and a mutation in gene **C** produces the lowest risk. However, only the best responses also referred to gene **D**. Students who did not mention any of the genes usually picked up one mark for noting that all of the mutant alleles increase the risk of lung cancer. Surprisingly, some thought that a mutation in gene **D** produces the highest risk.

(c)     Just fewer than 40% of students gave the correct answer of **180**.

(d)     Two-thirds of students scored at least two marks. Many were able to identify the decrease, plateau and increase for healthy cells and cancer cells. However, relatively few made reference to the plateau occurring for the same length of time. Students who failed to gain a mark for a similarity usually ignored the plateau. Most students spotted that a greater number of healthy cells were killed or that they experienced a faster decrease in number. Similarly, it was impressive to see that some used data from the graph to calculate that a greater *proportion* of cancer cells were killed. Many students also noted the faster increase in the number of healthy cells.

(e)     Half of students scored full marks. This was usually for mentioning that too many healthy cells would be killed, which could kill the patient or cause side effects. However, relatively few appreciated that it would take time to replace the healthy cells that had been killed.

**Q2.**

(a)     (i)      Over 90% of students correctly determined that base sequence could code for a maximum number of four amino acids.

(ii)     The vast majority of students gained at least one mark, often by mentioning a change in the sequence in amino acids. However, a significant number of students incorrectly referred to 'different amino acids being formed'. Most students gained a second mark for explaining that the active site/ tertiary structure would be altered. Over 50% of students gained maximum marks either by linking this to enzyme-substrate complexes not being formed or to changes in hydrogen bonds.

(b)     Most students had little difficulty in using the information to give two symptoms of phenylketonuria and gained both marks.

(c)     The majority of students obtained this mark, often by referring to migration or by describing interbreeding. However, over a third of students failed to gain credit and often accounted for the spread of phenylketonuria by horizontal or vertical gene transfer.

**Q3.**

(a)     Approximately two-thirds of students obtained at least one mark in this question, usually for mentioning that the cell-surface membrane has a phospholipid bilayer. Approximately one in four students also correctly interpreted that steroid hormones would be ‘lipid soluble’, enabling them to pass rapidly through the cell-surface membrane. A significant number of students simply referred to steroid hormones as being ‘fat soluble’, ‘non-polar’ or not being ‘water soluble’, responses which were not credited. Some students suggested that steroid hormones would pass through protein channels and/or carriers, or that active transport or water potential gradients were involved.

(b)     This question was generally well answered with almost 80% of students obtaining at least one of the two marks available. Most of these students referred to the complementary nature of the structures involved. Unfortunately, some students disqualified this point by referring to an active site or enzyme-substrate complex. Surprisingly, fewer students mentioned tertiary structure/shape, often simply indicating that the molecules were specific. Consequently, only 40% of students obtained both marks.

(c)     This was another question with a high discrimination index. The best responses included all information on the mark scheme which provided alternatives to access maximum marks. Nevertheless, only 54% obtained a mark and only half of these students obtained both marks, by showing a clear understanding of how AR could stimulate gene expression. Students obtaining a single mark often did so by describing that AR attaches to the promotor (region). Incorrect responses included references to DNA polymerase (rather than RNA polymerase), methylation, acetylation and histones.

(d)     This question was not well answered with less than 30% of students obtaining any of the three marks available. The main reason for this was that most students incorrectly concluded that the higher the number of CAG repeats, the greater the risk of developing prostate cancer. The probability value at ≤17 CAG repeats was often thought to be an anomaly or to show the highest risk of developing prostate cancer. There was considerable confusion and misuse of the terms ‘probability’, ‘chance’ and ‘risk’. The P values were frequently thought to show the probability of developing prostate cancer or to show the probability of having that number of CAG repeats. Very few students mentioned accepting or rejecting the null hypothesis and those who did sometimes had it the wrong way round. Some students thought that no statistical test had been carried out. Only 6% of students provided a suitably detailed conclusion to gain all three marks.

**Q4.**

(a)     The mark scheme for this question reflected the considerable variation in acceptable definitions for the term genome. Despite this, nearly 40% of students did not obtain the mark. There some very detailed definitions which often included the DNA in mitochondria and chloroplasts. The most common incorrect responses referred to “all the genes in a chromosome” or “all the genes in a species”.

(b)     Almost 80% of students obtained at least one mark for this question, often for referring to complementary structures. Almost half of these students gained a second mark by referring to a specific tertiary structure, shape or binding site. Weaker responses suggested that the term complementary is equivalent to ‘similar’ or referred to ‘active site’ rather than binding site.

(c)     This proved more difficult than expected with only 26% of students obtaining the mark. Incorrect responses included (unbound) DNA fragments, nucleotides, exons, introns, DNA without an antibody or transcription factor bound to it, and any combination of two of the three parts required to gain the mark. The constituent parts of a DNA molecule were also listed.

(d)     Again, relatively few students (30%) obtained the mark for this question. The most frequent correct responses referred to cDNA binding to the *P34* gene, preventing its transcription, or binding to mRNA, preventing its translation. The binding of the cDNA to mRNA, resulting in the destruction of mRNA, was also described. Binding to the promoter region was less frequently awarded. A significant minority of students mentioned that cDNA prevented RNA polymerase binding to the *P34* gene or just prevented its transcription, but with no suggestion of how. Incorrect responses often referred to cDNA increasing/decreasing methylation of the *P34* gene or simply that cDNA could not be transcribed into mRNA and translated to form the *P34* protein.

(e)     Considering that this question was relatively straightforward, it proved to be a very effective discriminator. Surprisingly, many students could not name both enzymes correctly. Consequently, only 35% of students obtained both marks for this question and 28% obtained a single mark. DNA helicase, reverse transcriptase and DNA polymerase were frequent incorrect responses. Even when the correct enzymes were named, students did not always describe the precise roles of the enzymes in the formation of recombinant plasmids. These descriptions were often too vague, e.g. “produces sticky ends” or “joins DNA together”, without any mention of the plasmid/vector. A significant minority referred to incorrect types of bonds when outlining the role of correctly named enzymes.

(f)      A significant number of students had difficulty applying their knowledge of electrophoresis to separating proteins. Only 14% of students gained both marks for this question, however 50% did at least obtain a single mark. Many students simply reverted to discussing DNA, especially VNTRs, or referred to DNA and protein, often suggesting that proteins consist of bases. Common responses which were not credited often referred to ‘size’ and ‘mass’, with no reference to the structure of proteins. However, better answers did obtain a mark when mass/size was linked to the number of amino acids or polypeptides. Difference in charge was the most frequently awarded marking point. Far fewer students mentioned different ‘R groups’ or ‘variable groups’.

**Q5.**

(a)     This was generally well answered, with over 40% of students obtaining both marks and over 80% gaining one mark. Most students stated that type II diabetics produce insulin, although a number of them simply stated that the pancreas is not damaged. The lack of responsiveness to insulin was not always linked to cells or receptors, or was described as a ‘resistance’ or ‘immunity’ to insulin. Although students often understood that diet and lack of exercise may be involved in causing type II diabetes, they did not always suggest that exercise and diet may be used in treating type II diabetes.

(b)     85% of students understood that insulin does not activate enzymes involved in the conversion of glycerol to glucose.

(c)     The idea of a transcription factor binding to a gene / DNA / promoter region, which then stimulates transcription by allowing RNA polymerase to bind, was clearly understood and described in the best responses. The inhibition of transcription by transcription factors was referred to less often. Over 50% of students gained at least one mark, often related to the binding process. Many students who did not gain credit limited their explanations to genes being switched on or expressed. Translation to form the proteins linked with pluripotency was often included in these answers. However, a number of students confused transcription and translation.

(d)     This question produced a good spread of marks and proved to be an effective discriminator. Over 90% of students gained at least one mark, and almost a third of students gained three out of the four marks available. Most students correctly compared the results of group A with group C. Fewer compared group A with group B. A significant minority of students mistakenly took group B results to be those of group A due to not carefully looking at the key provided for the graph; this restricted their maximum mark to two. Mice often became rats during the explanations but this was not penalised! Most students appreciated that the results could be different for humans, and stated that 12 weeks was not long enough to determine long-term effects. Some students referred to the lack of a statistical test, but this was not credited in this question. Similarly, for this investigation, thirty mice was not considered to be a small sample size.

**Q6.**

(a)     Over a third of students obtained both marks for this question, for answers of 19.41 / 19.4% or 19.47 / 19.5%, depending on whether the student used 7 x 52 (weeks) or 365 as the number of days in a year. Almost a third of students gained one mark for correctly calculating the increase in AD cases per year as being 1 048 320 or 1 051 200, depending on the number of days used. Incorrect rounding to give 19.46% was quite common, to gain one mark.

(b)     The majority of students gained at least one mark for stating that less acetylcholine would be broken down, or that more acetylcholine would be present. Almost half of these students obtained a second mark for stating that the acetylcholine binds to receptors. However, only 10% of students obtained maximum marks by describing how an impulse would be produced in the postsynaptic neurone. Many students did appreciate that sodium ion channels would open, but then failed to mention that sodium ions would then enter to cause depolarisation.

(c)     Over 50% of students scored zero for this question, usually due to answers lacking complete explanations for the valid suggestions they outlined. Less than 2% of students gained both marks. The most frequently credited response was that isolation had resulted in a small gene pool or low genetic diversity. Poor use of terminology also prevented many students gaining both marks. Invariably, students referred to the gene or mutation, rather than the allele being inherited from a (common) ancestor. There was also considerable confusion in the use of the terms inbreeding and interbreeding. There were also many responses which referred to genetic bottlenecks, the Founder effect, and an increase in the rate of mutation in isolated areas. A significant number of students suggested that the late onset of AD enabled individuals still to reproduce and pass on the mutation. This would explain why the frequency of the mutation had not been reduced (part d), rather than why there is a high frequency of this mutation to begin with.

(d)     Almost a third of students obtained both marks, clearly expressing the idea that, due to the symptoms of AD developing late on, affected individuals would have already reproduced. Over 50% of students scored zero, often providing responses that suggested that the mutation was not harmful, or indeed that it was beneficial. An improvement in health care was also provided as an explanation for the frequency of the mutation not being reduced. Students obtaining one mark often did not refer to the late onset of AD, but did understand that individuals with the mutation could still reproduce and pass on the allele.

(e)     Over 50% of students scored zero on this question. Many of these responses suggested that differences in the ‘level’ of acetylcholine, or exposure to mutagenic agents, caused the variation in the age at which the mutation is expressed. Over a third of students obtained one mark, usually by naming an environmental factor such as diet, smoking or stress. Answers specifically referring to epigenetics for at least one mark were infrequent. These responses often gained a second mark, 10% of students, for mentioning methylation or acetylation. Students who described these processes generally provided correct details.

(f)      Almost three out of ten students obtained one mark for explaining the sample size of 204 in terms of two copies of chromosome 14 or two copies of an allele. A common error was to refer to two chromatids. Explaining why there were only 74 potential AD cases when 75 mutations had been detected proved very challenging, with only 2% of students gaining this second mark. Many students suggested that the allele causing AD is recessive, despite line 18 of the comprehension passage stating that it is dominant. A common misconception was that one individual was heterozygous for the condition. Other incorrect responses focused on AD not having yet developed, or attempted to explain the data in relation to the degeneracy of the genetic code.

(g)     Over 10% of students did not attempt this question, and over 5% omitted the parts (e) and (f). It seems likely that some students had difficulty completing the paper, but it was also evident that these last three questions were demanding. Only 25% of students obtained a mark on this question. Almost all of these students gained one mark for realising that the GCA triplet would occur in a number of different places. Half of these students then explained that you could not then determine if the mutation was present or not. As in part (f), a number of incorrect ideas were linked to the degenerate nature of the genetic code. The misconception that probes were being used to sequence the whole genome arose, so lots of different primers would be needed and it would be very time consuming and costly. The probe was sometimes thought only to be able to identify the mutation if the gene had been expressed to cause the disease, or that the probe would not bind because the mutation had not occurred yet. A surprising number of students said the sequence of the mutation was not known, so therefore a probe could not be made, or that the mutation was different in different people.

**Q10.**

(a)     44% obtained one mark, usually for noting that binding of interferon gamma activated/switched on the enzyme inside the cell. 28% obtained a second mark, usually for reference to there being a second messenger process involved, or describing this. A few noted that ATP would be used in the phosphorylation of STAT1.

(b)     It was pleasing to find that 33% could correctly identify the transcription factors. Those who obtained one mark (42%) usually referred to STAT1, rather than phosphorylated STAT1 in their answer.

(c)     37% obtained two marks for reference to interferon gamma leading to more helper T cells, which then produced more interferon gamma. Some students (41%) only wrote about more of one or the other and scored 1. Poor answers usually consisted of generalised statements about, or definitions of, positive feedback.

(d)     70% of students obtained one mark, usually for stating that the *IRF* gene would slow/stop cell division; essentially stating what a tumour suppressor gene is. Only 20% obtained a second mark for noting that the *IRF* gene leads to the formation of IRF protein that binds to gene B. Very few went on to get the third mark by noting that this binding leads to the death of damaged or mutated (potential tumour) cells.

**Q11.**

(a)     The answers to this question and to Question 6 (b) suggested that many candidates were uncertain as to the roles of various enzymes used in gene technology.

(b)     Although the majority of candidates clearly understood the basic idea of transcription, they tended to gain limited credit for part (i) of this question. This was largely because they failed to make effective use of the material with which they had been provided. There were few references either to binding to the promoter or to stimulation of the enzyme. In part (ii), most candidates recognised the specificity of the receptors but were not always able to address the question of why oestrogen does not affect other cells. There was much discussion of oestrogen binding to receptors, but relatively little about receptors being confined to the cells of target organs.

(c)     Most candidates recognised the molecular structures as being either complementary or similar and used this information to construct sensible suggestions about reduction in growth of breast tumours. The terminology used by many, however, suggested confusion with enzyme action.

**Q12.**

(a)     Most candidates were able to explain that totipotent cells were able to differentiate but could not link this satisfactorily to the evidence in the table. Many of the less able candidates either attempted to link totipotency to callus, or considered callus, leaves and plantlets to be different sorts of cells.

(b)     Simple numbers and a generous mark scheme should have enabled most candidates to gain full credit for their answers to this question. The fact that only just over half did so emphasises the difficulty candidates have in working with ratios.

(c)     Part (i) was answered very poorly and many candidates ignored the reference to reproducing sexually in the question to write about mutation and environmental factors. Many of those who did approach the question appropriately demonstrated confusion between seeds and gametes and between mitosis and meiosis. The answers to part (ii) were rather better with many pointing out that plants derived from tissue culture would be clones or would be genetically identical. However, there were inappropriate responses relating to the procedure being “quicker” or “less expensive”.