HELICATE · A-LEVEL BIOLOGY · TOPIC BANK

3.8.2 Gene expression is controlled by a number of features

Mark scheme

Version 1 · 16 October 2026

TOTAL MARKS	QUESTIONS
83	11

How to use this mark scheme

;	Separates marking points. Each semicolon marks the end of one creditable point, and each point is worth one mark.
;;	Two marks for this answer (used for calculations).
/	Alternative words or phrases that are credited for the same point.
OR	An alternative answer for the same mark.
( )	Words in brackets are not needed to gain the mark.
max	More marking points are listed than marks available. Award up to the number of marks shown.
Accept	An alternative answer that gains credit.
Reject	A wrong answer. It does not gain credit and cancels a correct point it contradicts.
Ignore	Neither credited nor penalised. Mark the rest of the answer.
ECF	Error carried forward: credit correct working that uses an earlier wrong answer.
AO	Assessment objective: AO1 knowledge and understanding; AO2 applying knowledge; AO3 analysing, interpreting and evaluating.

Where an answer is correct but not listed, credit it if it is biologically accurate and answers the question at the depth the marks require. Right + wrong = wrong: a contradiction cancels a correct point.

Stem cells and cell specialisation 3.8.2.1

Question	Marking guidance	Mark	Comments
01.1	Type of stem cell: Totipotent Where it is found: Early embryo, for a limited time only Cell types it can produce: Any type of body cell;	**1** (1 × AO1)	Totipotent cells occur only for a limited time in early mammalian embryos and can produce any type of body cell.

Question	Marking guidance	Mark	Comments
01.2	1. (Cells) translate only part of their DNA OR only some genes are expressed/transcribed (and translated); 2. Different genes are expressed in different cells OR different cells translate different parts of the DNA; 3. So (only) certain proteins are made, which give the cell its specialised structure/function;	**2 max** (2 × AO1)	**Ignore** 'switched on/off'; the answer must refer to expression, transcription or translation **Reject** cells lose genes/DNA OR genes are removed 3. **Accept** named protein, eg enzymes, actin/myosin

Question	Marking guidance	Mark	Comments
01.3	1. From adult (somatic/body) cells OR from specialised cells, eg skin cells; 2. Using (appropriate protein) transcription factors OR by adding genes for transcription factors; 3. (These) cause transcription/expression of genes that make the cells pluripotent OR that allow the cells to divide (in unlimited numbers) and differentiate into other cell types;	**2 max** (2 × AO1)	2. **Accept** 'proteins that bind to the promoter/DNA and stimulate transcription' **Ignore** 'switched on' **Reject** embryonic cells as the source

Question	Marking guidance	Mark	Comments
02.1	Correct answer for 2 marks: 64.3 (%);; Accept for 1 mark: (28 − 10) ÷ 28 (× 100) OR 18 ÷ 28	**2** (2 × AO2)	**Accept** 64 or 64.29 for 2 marks Plotted values at week 12 are 28.0 and 10.0 mmol dm^{-3}. Accept readings within ±0.5 of each value, giving answers of 61.8–66.7%, for 2 marks **Reject** 35.7% (1.0×10^6 cells as a percentage of no cells) for any mark

Question	Marking guidance	Mark	Comments
02.2	1. The mouse's own cells have the same antigens/proteins (on their cell-surface membrane) OR are recognised as self; 2. So there is no immune response OR the transplanted cells are not rejected/destroyed (and keep secreting insulin); 3. Cells from a different mouse would be recognised as non-self/foreign and destroyed (by T cells/antibodies) OR would need immunosuppressant drugs;	**2 max** (1 × AO1) (1 × AO2)	2. **Accept** 'no rejection' **Ignore** 'the cells are compatible' unqualified **Ignore** 'same DNA' unless linked to antigens or rejection

Question	Marking guidance	Mark	Comments
02.3	1. Pluripotent cells can divide in unlimited numbers OR could divide uncontrollably; 2. So could form a tumour; 3. Could differentiate into other (unwanted) types of cell (in the pancreas/in the mouse) OR into cells that do not secrete insulin;	**2 max** (2 × AO2)	1. **Accept** mitosis for divide 1. **Ignore** 'grow uncontrollably' for divide uncontrollably **Accept** 'cancer' for tumour

Question	Marking guidance	Mark	Comments
02.4	1. (For:) only 2.0×10^6 cells brought glucose into the healthy range (6.0 mmol dm^{-3}, within 4.5–6.5); 2. (For:) glucose fell fastest/furthest with 2.0×10^6 cells (from about 25 to 7.4 by week 4); 3. (Against:) most tumours with 2.0×10^6 cells (3 of 10) OR tumours increase with the number of cells; 4. 1.0×10^6 cells lowered glucose a lot (to 10.0) with fewer tumours (1 of 10), so a smaller number/a number between 1.0 and 2.0×10^6 should be tested; 5. The check may have missed a few cells that were still pluripotent/undifferentiated, and more cells transplanted means more of these, so a better way of removing them is needed before using more cells; 6. Only 12 weeks, so tumours may develop later OR long-term control of glucose is unknown; 7. Mice, not humans, so the number of cells needed may be different (eg humans are larger); 8. Diabetes in these mice was caused by a chemical; in human type 1 diabetes, the (autoimmune) immune response could destroy some of the new insulin-secreting cells, so a different/larger number of cells may be needed;	**5 max** (5 × AO3)	To gain 5 marks, the answer must use both the glucose data and the tumour data 7. 'Mice, not humans' alone is not credited; it must be linked to the number of cells 8. Must be linked to the number of cells needed 1–4. **Accept** readings within ±0.5 mmol dm^{-3} of the plotted values **Ignore** sample size and statistical tests **Ignore** 'one study' or 'no repeats'

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Question	Marking guidance	Mark	Comments
03.1	1. Bleach kills microorganisms/bacteria/fungi (on the surface of the explants) that would grow on the nutrient agar OR that would compete with/damage the explant; 2. Rinsing removes the bleach, which would kill/damage the plant cells/explant; 3. The water is sterile so no microorganisms are added back (after the bleach);	**2 max** (2 × AO2)	1. **Accept** 'sterilises/disinfects the explants' only if linked to growth of microorganisms or contamination of the culture **Ignore** 'germs' **Reject** 'kills viruses' alone

Question	Marking guidance	Mark	Comments
03.2	Correct answer for 2 marks: 84 (%);; Accept for 1 mark: 31 ÷ 37 (× 100) OR 83.8	**2** (2 × AO2)	**Reject** 77.5 or 78% (31 ÷ 40) for any mark

Question	Marking guidance	Mark	Comments
03.3	1. Cells in the floret tips are unspecialised and can (still) divide (by mitosis) and differentiate; 2. Different cells express different genes/translate different parts of their DNA, so they become root cells or shoot cells; 3. Stalk cells are (already) specialised OR express/translate only the genes/part of the DNA needed for their (stalk) function;	**2 max** (1 × AO1) (1 × AO2)	**Ignore** 'totipotent' or 'stem cells' unqualified **Ignore** 'switched on/off' **Reject** stalk cells have lost genes/DNA

Transcription factors and oestrogen 3.8.2.2

Question	Marking guidance	Mark	Comments
04.1	They move from the cytoplasm into the nucleus and can stimulate or inhibit transcription;	**1** (1 × AO1)	

Question	Marking guidance	Mark	Comments
04.2	1. Oestrogen binds to a receptor (site) on a transcription factor (in the cytoplasm); 2. This changes the shape of the transcription factor OR activates the transcription factor OR releases it from an inhibitor; 3. The transcription factor moves from the cytoplasm into the nucleus (through a nuclear pore); 4. The transcription factor binds to the promoter (region) of the target gene; 5. (This) stimulates RNA polymerase (to bind) OR allows RNA polymerase to begin transcription;	**4 max** (4 × AO1)	1. **Accept** 'oestrogen binds to its receptor, which is a transcription factor'; accept '(inactive) transcription factor' for receptor **Reject** active site, enzyme-substrate complex or induced fit for the receptor, once only 4. **Accept** 'specific base sequence of the DNA' for promoter; ignore 'binds to the gene/bases' unqualified 5. **Reject** DNA polymerase **Ignore** methylation, acetylation or histones

Question	Marking guidance	Mark	Comments
04.3	Correct answer for 2 marks: 82.8 (%);; Accept for 1 mark: (6.4 − 1.1) ÷ 6.4 (× 100) OR 5.3 ÷ 6.4	**2** (2 × AO2)	**Accept** 83 or 82.81 for 2 marks Plotted means are 6.4 and 1.1. Accept readings within ±0.1 of each value (6.3–6.5 and 1.0–1.2), giving answers of 81.0–84.6%, for 2 marks **Reject** 17.2% (oestrogen + R as a percentage of oestrogen) for any mark

Question	Marking guidance	Mark	Comments
04.4	1. R binds to the oestrogen receptor/transcription factor (at the same site) so oestrogen cannot bind; 2. R does not activate the receptor/transcription factor OR does not change its shape, as R alone gives a similar result to no addition; 3. So the transcription factor does not (move into the nucleus and) bind to the promoter (of gene H) OR RNA polymerase is not stimulated; 4. So little transcription of gene H OR less mRNA (1.1 compared with 6.4);	**3 max** (3 × AO2)	1. **Accept** '(inactive) transcription factor' for receptor 1. 'Competitive inhibitor' alone is not enough; the answer must say R binds to the receptor **Reject** R binds to oestrogen **Reject** active site or enzyme-substrate complex, once only **Ignore** 'switched off'

Question	Marking guidance	Mark	Comments
04.5	1. (Supports:) the standard deviations for oestrogen + R overlap with those for no addition and for R only, so there is no significant difference (between them) OR the differences are likely to be due to chance; 2. (Against:) the mean with oestrogen + R is (slightly) higher than with no addition or with R only (1.1 compared with 1.0 and 0.8), so R might not prevent all the effect; 3. Only one concentration of oestrogen/R was tested OR a higher concentration of oestrogen might overcome R; 4. mRNA was measured only after 24 hours, so R might only delay the effect of oestrogen;	**2 max** (2 × AO3)	1. **Accept** SDs; accept 'error bars overlap' **Reject** 'the results are significant' unqualified

Epigenetics and RNA interference 3.8.2.2

Question	Marking guidance	Mark	Comments
05.1	Increased methylation of the DNA of the gene;	**1** (1 × AO1)	

Question	Marking guidance	Mark	Comments
05.2	1. Histones bind (more) tightly to the DNA OR chromatin is more condensed/tightly packed; 2. Transcription factors/RNA polymerase cannot bind to the promoter/DNA (of the gene);	**2** (2 × AO1)	1. **Accept** 'DNA is less accessible' 2. **Accept** 'promoter (region)' for DNA **Reject** acetylation of the DNA **Ignore** 'switched off'

Question	Marking guidance	Mark	Comments
06.1	1. Identical twins have the same DNA base sequence OR the same genes/alleles OR are genetically identical; 2. So differences in methylation (between them) are not due to genetic factors OR must be due to (differences in) the environment;	**2** (2 × AO2)	1. **Accept** 'same genotype/genome' 2. **Accept** 'due to environment or chance' **Reject** 'identical twins have the same methylation'

Question	Marking guidance	Mark	Comments
06.2	1. Young identical twins differ less than young non-identical twins (1.8 compared with 7.4), so genetic factors influence methylation; 2. Older identical twins differ more than young identical twins (6.3 compared with 1.8), so differences build up with age/with time in different environments; 3. Identical twins who lived apart differ more than those who lived together (10.5 compared with 6.3), so the environment influences methylation; 4. Older identical twins who lived apart differ more than young non-identical twins (10.5 compared with 7.4), so over a lifetime the environment can have a larger influence than genetic differences; 5. Only means given OR no standard deviations/statistical test, so we cannot tell whether differences are significant OR only 12 pairs lived apart; 6. No data for older non-identical twins OR for young identical twins who lived apart;	**4 max** (4 × AO3)	**Accept** correct manipulation of the data, eg 6.3 is 3.5 times 1.8 **Max 3** if no data from Table 3 are used **Ignore** 'age causes methylation' unqualified **Reject** 'methylation changes the base sequence' OR 'methylation is a mutation'

Question	Marking guidance	Mark	Comments
07.1	The same volume of solvent (without drug V);	**1** (1 × AO3)	**Accept** 'solvent only' **Ignore** reasons, eg 'to show the solvent has no effect' **Reject** 'water' or 'nothing'

Question	Marking guidance	Mark	Comments
07.2	1. Drug V prevents removal of acetyl groups OR increases/maintains acetylation of histones (associated with gene W); 2. Histones bind less tightly to the DNA OR chromatin is less condensed; 3. Transcription factors/RNA polymerase can bind to the promoter (of gene W); 4. More transcription of gene W (mRNA rises from 1.0 to 4.9); 5. More tumour suppressor protein is made (by translation), which slows/controls cell division; 6. So fewer cells as the concentration of drug V increases (52 to 16 × 10^4);	**4 max** (2 × AO1) (2 × AO2)	**Max 3** if no data from Table 4 are used 1. **Reject** acetylation of DNA; ignore methylation of histones 3. **Accept** 'promoter (region)' OR DNA for promoter 4. **Accept** 'gene W is expressed'; ignore 'switched on' 5. **Ignore** 'growth' for cell division **Reject** any reference to mutation in line P

Question	Marking guidance	Mark	Comments
07.3	Correct answer for 2 marks: 65.4 (%);; Accept for 1 mark: (52 − 18) ÷ 52 (× 100) OR 34 ÷ 52	**2** (2 × AO2)	**Accept** 65 or 65.38 for 2 marks **Reject** 34.6% (cells at 2.0 as a percentage of no drug) for any mark

Question	Marking guidance	Mark	Comments
07.4	1. Drug V still increased transcription of gene W in line Q (mRNA 1.0 to 4.8); 2. The deletion changes the amino acid sequence/causes a frame shift, so the tumour suppressor protein is non-functional OR has a different tertiary structure; 3. So cell division is not slowed OR cell number hardly changes (55 to 51 × 10^4);	**2 max** (1 × AO2) (1 × AO3)	**Max 1** if point 2 is not given 2. **Accept** 'no functional tumour suppressor protein is made' **Reject** 'drug V did not affect line Q' for point 1 **Ignore** 'switched on'

Question	Marking guidance	Mark	Comments
07.5	1. Take a sample of the tumour cells and find the base sequence of gene W OR test gene W for mutations; 2. Patients whose gene W has a normal base sequence are likely to benefit, (like line P) because the drug can restore production of functional tumour suppressor protein; 3. Patients with a mutated gene W are unlikely to benefit, (like line Q) because more transcription still gives non-functional protein; 4. Check that gene W is rarely transcribed/has little mRNA OR that histone acetylation (at gene W) is low in the tumour cells, so the cause is epigenetic; 5. Grow the patient's tumour cells with drug V and see whether cell number falls;	**3 max** (3 × AO3)	1. **Accept** 'genetic test/screen the tumour for the gene W mutation' **Ignore** 'test the drug on the patient first' **Reject** 'test for histone deacetylase in the blood'

Question	Marking guidance	Mark	Comments
08.1	1. The (single-stranded) RNA has a base sequence complementary to (part of) the mRNA for protein K; 2. (So) it binds to/base pairs with the mRNA (forming double-stranded RNA); 3. Ribosomes cannot bind/translate the mRNA OR the mRNA is broken down, so (less) translation;	**2 max** (2 × AO1)	**Reject** binds to the gene/DNA **Reject** 'prevents transcription' **Reject** 'translation of the gene' 3. **Accept** 'less protein K is made' only if linked to translation of mRNA

Question	Marking guidance	Mark	Comments
08.2	Correct answer for 2 marks: 75 (%);; Accept for 1 mark: (96 − 24) ÷ 96 (× 100) OR 72 ÷ 96	**2** (2 × AO2)	Plotted means are 96 and 24. Accept readings within ±1 of each value (95–97 and 23–25), giving answers of 73.7–76.3%, for 2 marks 72 on its own (the difference in percentage points) = 0 marks **Reject** 25% for any mark

Question	Marking guidance	Mark	Comments
08.3	1. (For:) the mean is lower at 0.6 mg kg^{-1} (18% compared with 24%); 2. (Against:) the standard deviations overlap, so there is no significant difference OR the difference is likely to be due to chance; 3. A higher dose may cause more side effects OR costs more (for no significant benefit);	**2 max** (2 × AO3)	2. **Accept** SDs; accept 'error bars overlap' **Reject** 'the results are not reliable' for point 2 **Ignore** sample size

Question	Marking guidance	Mark	Comments
08.4	1. The RNA (from the drug) is broken down OR is not replaced; 2. The base sequence of the gene (for protein K) is not changed, so it is still transcribed OR mRNA is still produced; 3. So the mRNA is translated (again) and protein K is made;	**2 max** (1 × AO1) (1 × AO2)	**Ignore** 'switched back on' **Reject** 'the gene is translated' **Reject** 'the drug caused a mutation that was repaired'

Gene expression and cancer 3.8.2.3

Question	Marking guidance	Mark	Comments
09.1	Type of tumour: Benign Rate of growth: Slow Appearance of cells: Similar to normal cells Spreads to other parts of the body: No;	**1** (1 × AO1)	Benign tumours grow slowly, their cells are similar to normal cells and they do not spread. Malignant tumours grow quickly, have unspecialised/abnormal cells and spread (metastasis).

Question	Marking guidance	Mark	Comments
09.2	1. More oestrogen binds to receptors/transcription factors (in the breast cells); 2. More transcription/expression of genes that stimulate cell division; 3. Rapid/uncontrolled cell division (forms a tumour);	**2 max** (1 × AO1) (1 × AO2)	3. **Accept** mitosis for cell division; reject meiosis **Ignore** 'growth' for cell division **Ignore** 'switched on'

Question	Marking guidance	Mark	Comments
10.1	1. Increased methylation of the promoter of gene T (4% to 78%); 2. Methylation prevents transcription factors/RNA polymerase binding to the promoter (of gene T); 3. So (much) less transcription of gene T (mRNA 1.00 to 0.08) OR less tumour suppressor protein made; 4. Tumour suppressor protein (normally) slows/controls cell division; 5. Change in base sequence of gene G (mutation) changes the amino acid sequence/primary structure of protein G; 6. So the tertiary structure/shape of protein G changes, so it is active without the growth factor; 7. Gene G has become an oncogene; 8. Amount of mRNA from gene G is (almost) unchanged (1.00 and 1.05), so the effect is due to the changed protein, not more transcription; 9. Rapid/uncontrolled cell division (mitosis) forms a mass of (abnormal) cells/tumour;	**6 max** (3 × AO1) (3 × AO2)	**Max 4** if only gene T or only gene G is discussed 1–3. **Accept** DNA/gene for promoter **Reject** 'methylation causes a mutation' OR 'methylation changes the base sequence' 5. **Accept** 'substitution mutation'; reject 'different amino acids are formed' 6. **Reject** oncogene/protein G is not made or is non-functional 7. **Ignore** proto-oncogene; a correct oncogene point next to it still scores 9. **Ignore** 'growth' for cell division; reject meiosis **Ignore** 'switched on/off'

Question	Marking guidance	Mark	Comments
10.2	1. In the first tumour, (permanently active) protein G (from the oncogene) causes the rapid division, so blocking it slows/stops division; 2. In the second tumour, the cause is a lack of tumour suppressor protein OR protein G is normal (only active with growth factor), so blocking protein G does not restore control of division; 3. A drug that inhibits/reduces methylation (of the promoter of gene T) OR inhibits the enzyme that methylates DNA; 4. So gene T is transcribed OR tumour suppressor protein is made (and cell division is controlled);	**3 max** (1 × AO1) (2 × AO2)	Points 3 and 4 may be credited for a correct answer without points 1 and 2 3. **Accept** 'demethylating drug' **Reject** any reference to mutation for points 3–4 **Ignore** 'switched on'

Question	Marking guidance	Mark	Comments
11.1	1. Probability of 0.003/0.3% that the difference (in risk from the reference group) is due to chance; 2. Less than 0.05/5%, so the difference is significant OR the null hypothesis is rejected;	**2** (2 × AO2)	1. **Accept** '99.7% probability that the difference is not due to chance' **Reject** '0.3% chance of getting cancer' 2. **Reject** 'the results are significant' unqualified

Question	Marking guidance	Mark	Comments
11.2	1. (Against:) the fast allele with little red meat gave no significant increase in risk (1.09, *P* = 0.48, more than 0.05); 2. (For:) the fast allele with more than 100 g red meat gave a significantly higher risk (1.87, 87% higher, *P* = 0.003); 3. So the risk depends on both the allele and diet OR on an interaction of genetic and environmental factors; 4. A lot of red meat with two slow alleles gave no significant increase (1.18, *P* = 0.21); 5. Correlation does not show causation OR other factors may differ between the groups, eg smoking/alcohol/fibre in the diet; 6. Diet recorded only at the start OR diet was self-reported, so may be inaccurate/may have changed over 12 years; 7. No data for people eating 50–100 g per day;	**4 max** (4 × AO3)	**Max 3** if only points from the data (points 1–4) or only limitations (points 5–7) are given **Ignore** sample size; the number of people is given **Ignore** length of investigation and use of a statistical test **Reject** 'P value is the chance of getting cancer'

Question	Marking guidance	Mark	Comments
11.3	1. Test/screen people (eg DNA) to find who has a fast allele; 2. Advise people with a fast allele to eat less red meat OR to avoid red meat cooked at high temperatures; 3. Offer people with a fast allele (who eat a lot of red meat) regular screening for colorectal cancer, so tumours are found early;	**2 max** (2 × AO2)	2. **Accept** 'change their diet' only if linked to red meat **Ignore** 'gene therapy'

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