M1.(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]

M2.(a) Translation.

1

(b) Transfer RNA / tRNA.

1

(c) TAC;

UAC.

2

(d) Have different R group.

Accept in diagram

1

(e) 1. Substitution would result in CCA / CCC / CCU;
2. (All) code for same amino acid / proline;
3. Deletion would cause frame shift / change in all following codons / change next codon from UAC to ACC.

3

[8]

M3.(a) 1. Helicase;
2. Breaks hydrogen bonds;
3. Only one DNA strand acts as template;
4. RNA nucleotides attracted to exposed bases;
5. (Attraction) according to base pairing rule;
6. RNA polymerase joins (RNA) nucleotides together;
7. Pre-mRNA spliced to remove introns.

6 max

(b) 1. Polymer of amino acids;
2. Joined by peptide bonds;
3. Formed by condensation;
4. Primary structure is order of amino acids;
5. Secondary structure is folding of polypeptide chain due to hydrogen bonding;

Accept alpha helix / pleated sheet

6. Tertiary structure is 3-D folding due to hydrogen bonding and ionic / disulfide bonds;
7. Quaternary structure is two or more polypeptide chains.

5 max

(c) 1. Hydrolysis of peptide bonds;
2. Endopeptidases break polypeptides into smaller peptide chains;
3. Exopeptidases remove terminal amino acids;
4. Dipeptidases hydrolyse / break down dipeptides into amino acids.

4

[15]

M4.(a) 1. Reduction in ATP production by aerobic respiration;
2. Less force generated because fewer actin and myosin interactions in muscle;
3. Fatigue caused by lactate from anaerobic respiration.

3

(b) Couple **A**,
1. Mutation in mitochondrial DNA / DNA of mitochondrion affected;
2. All children got affected mitochondria from mother;
3. (Probably mutation) during formation of mother's ovary / eggs;

Couple **B**,
4. Mutation in nuclear gene / DNA in nucleus affected;
5. Parents heterozygous;
6. Expect 1 in 4 homozygous affected.

4 max

(c) 1. Change to tRNA leads to wrong amino acid being incorporated into protein;
2. Tertiary structure (of protein) changed;
3. Protein required for oxidative phosphorylation / the Krebs cycle, so less / no ATP made.

3

(d) 1. Mitochondria / aerobic respiration not producing much / any ATP;
2. (With MD) increased use of ATP supplied by increase in anaerobic respiration;
3. More lactate produced and leaves muscle by (facilitated) diffusion.

3

(e) 1. Enough DNA using PCR;
2. Compare DNA sequence with 'normal' DNA.

2

[15]