

No part of the candidate evidence in this exemplar material may be presented in an external assessment for the purpose of gaining credits towards an NCEA qualification.

2

91159



911590



NEW ZEALAND QUALIFICATIONS AUTHORITY  
MANA TOHU MĀTAURANGA O AOTEAROA

SUPERVISOR'S USE ONLY

## Level 2 Biology, 2017

### 91159 Demonstrate understanding of gene expression

2.00 p.m. Wednesday 22 November 2017

Credits: Four

| Achievement                                   | Achievement with Merit                                 | Achievement with Excellence                                 |
|-----------------------------------------------|--------------------------------------------------------|-------------------------------------------------------------|
| Demonstrate understanding of gene expression. | Demonstrate in-depth understanding of gene expression. | Demonstrate comprehensive understanding of gene expression. |

Check that the National Student Number (NSN) on your admission slip is the same as the number at the top of this page.

**You should attempt ALL the questions in this booklet.**

If you need more space for any answer, use the page(s) provided at the back of this booklet and clearly number the question.

Check that this booklet has pages 2–12 in the correct order and that none of these pages is blank.

**YOU MUST HAND THIS BOOKLET TO THE SUPERVISOR AT THE END OF THE EXAMINATION.**

Merit

TOTAL

13

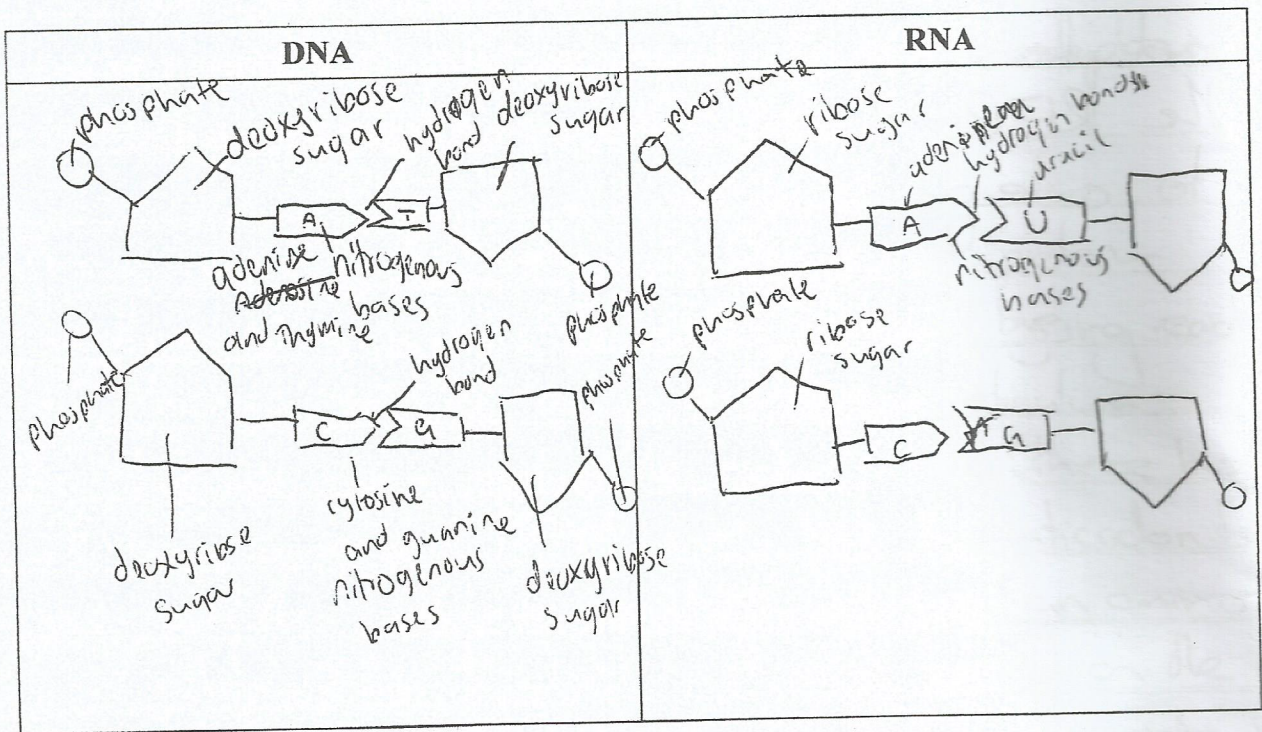
ASSESSOR'S USE ONLY

## QUESTION ONE: PROTEIN SYNTHESIS

- (a) In the table below, draw a DNA and an RNA molecule, each composed of the FOUR different nucleotides that are specific to each molecule.

In your answer you **must** include and label where appropriate:

- phosphate
- sugar (deoxyribose or ribose)
- nitrogenous bases (adenine, cytosine, guanine, thymine, and uracil)
- hydrogen bond.



- (b) Discuss the relationship between DNA, mRNA, and tRNA in protein synthesis.

In your answer include:

- an explanation of the key stages of protein synthesis
- an explanation of why tRNA is shorter than mRNA, when considering their function
- a discussion, with two justified reasons, why DNA is not directly translated into a polypeptide chain.

DNA is a double stranded molecule containing genetic information. In the key stages of protein synthesis, it starts with transcription. DNA must be copied identically so it can leave the nucleus to undergo translation, but it cannot as it is double stranded.



So a single strand (the template strand) is used to be copied onto mRNA strand which is only a single strand so it can leave the nucleus. DNA ~~is unzipped~~ begins transcription by using a promoter region which enables ~~DNA~~ RNA polymerase to start <sup>transcribing</sup> ~~copying~~ the ~~DNA~~ template strand onto the mRNA. Once the RNA polymerase enzyme reaches a terminator region it stops <sup>transcribing</sup> ~~copying~~ the DNA. The mRNA strand is identical to the <sup>coding</sup> ~~template~~ strand apart from <sup>changing</sup> ~~switching~~ the ~~base~~ nucleotide base of thymine to uracil. According to base pairing rules, ~~adenosine~~ adenine now bonds with hydrogen bonds to ~~th~~ uracil and guanine to cytosine. The groups of 3 bases are called codons on the mRNA rather than triplets on the DNA strand. The mRNA strand leaves the nucleus through nuclear pores and into the cytoplasm in order to start translation. A ribosome will attach to the mRNA strand and begin translation. tRNA molecules attach to amino acids and join by their corresponding anti-codons to the <sup>complementary</sup> ~~codon~~ sequence on the mRNA, <sup>according to base pairing rules</sup> building a chain of amino acids as more ~~one~~ anticodons and codons are bonded.

Amino acids are a monomer, proteins are a polymer made

There is more space for your answer to this question on the following page



up of many repeating units of amino acids. The amino acids bond by ~~the~~ peptide bonds to form a polypeptide chain and therefore a protein. tRNA is shorter than mRNA as mRNA contains all the codons on the strand, whereas the tRNA is only bringing one anti-codon per molecule to ~~join~~ bond to the mRNA strand. DNA is not directly translated into ~~proteins~~ <sup>or polypeptide chain</sup> as it is a double stranded molecule and double stranded molecules are too big to leave the nucleus, which must occur for translation to occur. Therefore, a single strand of mRNA is formed to leave and be translated because the DNA cannot. It also cannot be ~~translated~~ directly translated into ~~proteins~~ as a polypeptide chain as some proteins are made into molecules like enzymes, which the body needs many of the same molecule of and if the DNA is removed from the nucleus and translated directly into a polypeptide chain it will not be able to create another identical enzyme or protein as the genetic material has been used. This is why the mRNA strand is used rather than directly translating the DNA into a polypeptide chain.



## QUESTION TWO: GENETIC CODE

 ASSESSOR'S  
USE ONLY

### mRNA (codon) : Amino Acid Table



Tracey Greenwood, Richard Allan, *Year 12 Biology 2003*, (Hamilton: Biozone, 2003), p 287.

- (a) A point mutation on the haemoglobin  $\beta$  gene can cause sickle cell disease. The template DNA sequence for part of the normal and mutated haemoglobin protein is shown in the table below. The affected base is shown in red, and indicated with an arrow.

Complete the normal and mutated amino acid sequence using the mRNA : Amino Acid table above.

|                     | Normal                  | Mutation causing sickle cell disease |
|---------------------|-------------------------|--------------------------------------|
| DNA template strand | GAC TGA GGA CTC AAC     | GAC TGA GGA CAC AAC                  |
| mRNA strand         | CUG ACU CCU GAG UUC     | CUG ACU CCU GUG UUC                  |
| amino acid sequence | Leu, Thr, Pro, Glu, Phe | Leu, Thr, pro, val, phe              |



(b) Discuss the effects of point mutations on final protein structure.

In your answer include:

- identification and a description of the type of mutation leading to sickle cell disease
- an explanation of how this mutation affects the amino acid sequence and final protein structure
- a discussion of how the degeneracy of the code can reduce the impact of point mutations on final protein structure, and on an organism's survival.

The type of mutation occurring in sickle cell disease is a substitution mutation as the base in the 4th triplet 'A' has been replaced with 'T'. This type of mutation can have an effect on the organism as it has changed the amino acid coded for from 'Glu' to 'Val', but may not have as large an effect on the organism as a insertion or deletion mutation as they can change the entire base sequence due to a reading frame shift. This mutation affects the amino acid sequence and final protein structure as it has changed the base sequence to code for a 'Val' amino acid rather than a 'Glu' amino acid. ~~Factors~~ TO ~~affect~~ synthesise protein structure amino acids are used in type of amino acid that they are and sequence of amino acids to code for the protein. The type of amino acid in the chain has been changed by this mutation therefore it can have an effect on the protein structure.

Humans only need 20 amino acids to make a protein but there are

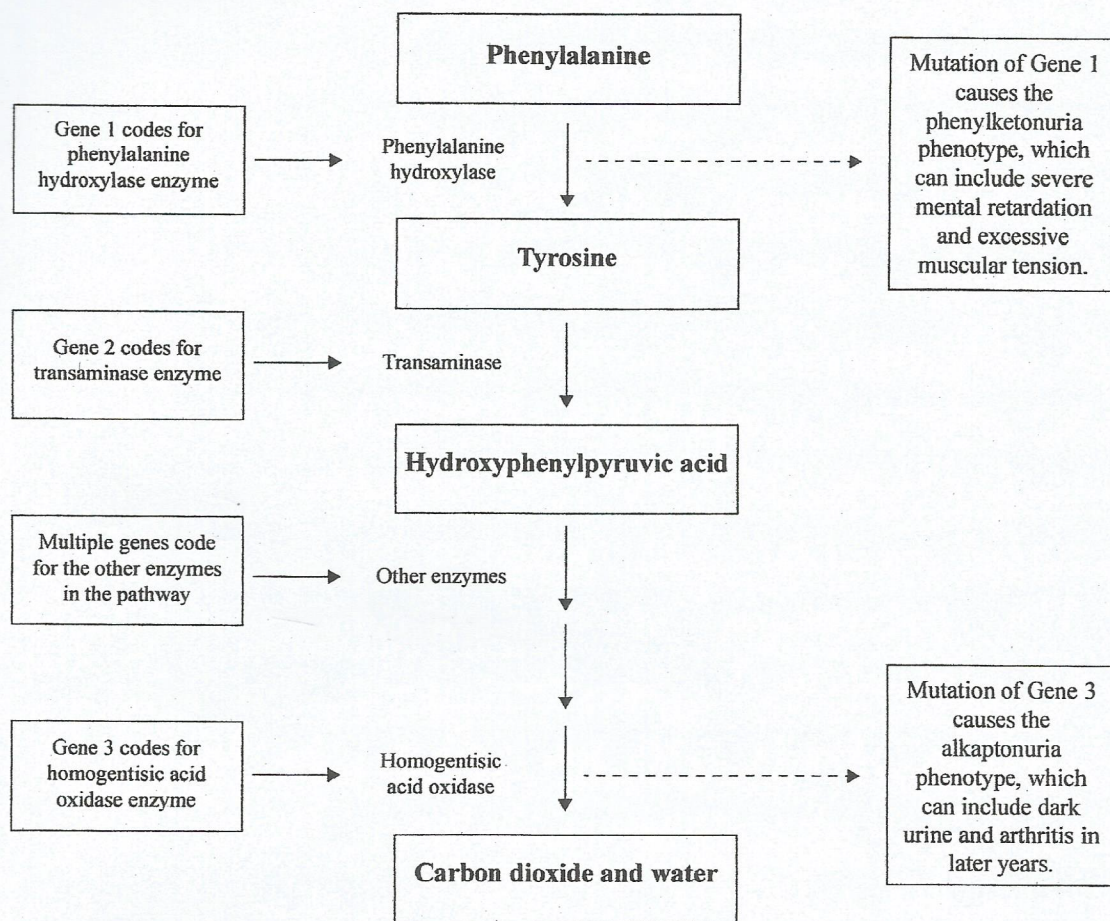


but possible combinations of bases in the codons. Because humans only need 20, some amino acids have slightly different bases but code for the same amino acid. For example, GUU, GUC, GUA, GUG all code for the amino acid 'val', even though they all have a different third base. This is the redundant code, due to the degeneracy of this code, if a substitution mutation substituted the third base of an amino acid, it would typically have little effect as many codons with differences in only their third base still code ~~for~~ for the same amino acid, and therefore will not have as great an effect on the protein as the type of amino acid coded for is still the same. This redundancy in the code reduces the impact of substitution mutations on final protein structure as the protein would still contain the same amino acids, and therefore would code to be the same protein, and reduces the impact on an organism's survival as the protein still functions as it would so there would be no effect on the organism from this mutation.



### QUESTION THREE: METABOLIC PATHWAYS

A simplified section of the phenylalanine metabolic pathway is shown below.



Using the simplified section of the phenylalanine metabolic pathway, discuss how the presence or amount of a product affects the phenotype.

In your answer:

- describe how enzymes control metabolic pathways
- explain the relationship between genes, enzymes, and products
- identify which mutation causes the more severe phenotype AND discuss how mutations affect the presence or amount of products in the phenylalanine metabolic pathway.

You may draw on the diagram above.

Enzymes control metabolic pathways as they are the things that change (add to, pull apart, alter) substances. Enzymes are a specific shape made for doing specific jobs and performing specific actions with specific molecules and substances. (the substance enters or sits in the the enzymes active point and is either added to, broken apart or changed in some way.)



in metabolic pathways, the substances need to go through multiple stages (they will be changed by many enzymes ~~multi~~ multiple times ~~at diff~~ along different stages of ~~the~~ the pathway and will create new substances that will be passed onto the next stage to be altered again ~~at~~ until the product desired is formed.). Genes are sections of DNA coding for different things. Genes can code for enzymes (eg: Gene 1 codes for phenylalanine hydroxylase enzyme); and these enzymes that the genes code for are used to change substances to create products. The substances react (~~are changed by~~) ~~the~~ with (are changed by) the enzymes, to create new substances that can be used in the next stage with the next enzyme. When the ~~1~~ different enzymes change the different substances or molecules, there is a final product made (after all the stages).  
is the final product.

The ~~most~~ mutation with the most severe phenotype ~~&~~ is the mutation of gene 1 as it ~~can~~ causes the phenylketonuria phenotype which can include severe mental retardation (slowing) and excessive muscular tension. (Where as the other mutation (of gene 3) ~~only~~ <sup>only</sup> causes alkaptonuria phenotype including which can include dark urine and arthritis later on in life, ~~which~~ These phenotypes / ~~&~~ symptoms are not as severe as mental retardation and excess muscular tension.) As well as Gene 1 having a more severe

There is more space for your answer to this question on the following pages.



phenotype, it also occurs at the beginning of the metabolic pathway. This can disturb the entire rest of the pathway. As specific enzymes need to be made ~~to be used in~~ ~~metabolic~~ to create specific substances/products, a mutation of this <sup>first</sup> enzyme will lead to an incorrect substance/product being made. Even if the rest of the pathway and enzymes are correct, they cannot work without the presence ~~of~~ of the substance of the previous stage. The mutation of the previous stage means incorrect products will be made.

As enzymes have a specific shape, if an incorrect substance/molecule tries to fit into its active site, the enzyme's shape can be altered or destroyed; meaning that now, not even the correct substances can use the enzyme. In the Phenylalanine metabolic pathway, the mutation at gene ~~one~~ 1 means that the needed Tyrosine cannot be made. The phenylalanine cannot be changed to Tyrosine product by phenylalanine hydroxylase enzyme ~~as~~ as it is altered or incorrect. ~~There~~ There will be very little product formed, or none at all (not that is correct).

\* This means that the following stages of the metabolic pathway cannot occur or will occur incorrectly.



| <b>Subject:</b> | <b>BIOLOGY</b>     | <b>Standard:</b>                                                                                                                                                                                                                                                                                                    | <b>91159</b> | <b>Total score:</b> | <b>13</b> |
|-----------------|--------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------|---------------------|-----------|
| <b>Q</b>        | <b>Grade score</b> | <b>Annotation</b>                                                                                                                                                                                                                                                                                                   |              |                     |           |
| 1               | M6                 | Anti-parallel nature and base pairing rule shown.<br>Unclear about transcription and translation.<br>Comprehensive explanation of Protein Synthesis is given.<br>Roles of tRNA, mRNA are explained<br>Explanation for tRNA being shorter than mRNA is given.<br>Reason for DNA not being directly translated given. |              |                     |           |
| 2               | A4                 | Identifies mutation as substitution, states a different amino acid is coded for and therefore the protein has a different structure.<br>Describes degeneracy.                                                                                                                                                       |              |                     |           |
| 3               | A3                 | States that genes code for enzymes.<br>States that a mutation stops the desired products forming and build up of products occurs.<br>Identifies that Gene1's mutation is more serious than that of Gene 3.                                                                                                          |              |                     |           |