# 2018 H2 A level Paper 2

Answer **all** questions.

- **1** Fig. 1.1 represents the molecular structure of a G-protein linked receptor in the cell surface membrane.
- (a) Discuss how the arrangement of molecules in Fig. 1.1 supports the fluid mosaic model of the cell membrane. [3]
  - It is referred to as 'fluid" because the cell membrane comprises of <u>phospholipids</u>\* and a <u>G-protein linked receptor</u>\* which are <u>free to move laterally</u> within a layer;
  - Presence of <u>cholesterol</u>\* molecules within each phospholipid layer <u>increases the fluidity</u> of the membrane;
  - 3. It is referred to as 'mosaic" because the <u>random arrangement</u> of the <u>proteins embedded</u> <u>amongst the phospholipid molecules</u> resemble a mosaic pattern;

## Teacher's comment:

It is insufficient to state that proteins and lipids are components of the membrane. How they are arranged must also be stated. Using descriptions like the proteins and phospholipids are arranged like 'mosaic tiles' is insufficient to gain any marks.

- (b) Explain how the molecular structure of the protein shown in Fig. 1.1 enables it to function as a G-protein linked receptor. [3]
  - G protein linked receptor (GPCR) a <u>seven pass transmembrane protein</u> consisting of <u>7 α-</u> <u>helices\*</u> connected by three intracellular and three extracellular peptide loops.
  - 2. The <u>intracellular domain</u>/cytoplasmic side of GPCR has a <u>*G* protein binding site</u>\* that allows binding of a heterotrimeric G protein complex.
  - 3. The <u>extracellular</u> loops has a *ligand binding site*\* at which a *specific*\* signaling molecule can bind to the GPCR.
  - 4. When a ligand binds to the ligand binding site at the extracellular side of a GPCR it causes a <u>conformational change</u>\* of the intracellular domain /at the cytoplasmic side of the GPCR,
  - 5. The activated GPCR can then <u>activate</u> an associated <u>G protein by exchanging its bound GDP</u> <u>for a GTP</u>.
- (c) A large range of stimuli can trigger the activation of G-proteins through G-protein linked receptors. These stimuli can include light, calcium ions and hormones such as glucagon.

State the common effect that these diverse stimuli have on G-protein linked receptors. [1]

- 1. The stimuli can trigger a <u>conformational change</u>\* in the intracellular domain /at the cytoplasmic side of the GPCR, activating the receptor.
- (d) Explain how the activation of the G-protein by the binding of glucagon to the G-protein linked receptor triggers downstream signalling pathways that result in a cascade of enzyme-catalysed reactions. [3]
  - 1. When glucagon binds a G-protein linked receptor (GPCR), the GPCR will undergo a <u>conformation change</u> in intracellular domain allowing G-protein to bind to it; and
  - 2. G-protein is activated when it <u>displaces its attached GDP</u> for GTP;
  - 3. Activated G-protein will <u>translocate along membrane</u> and <u>bind to enzyme adenylyl</u> <u>cyclase and phosphorylate it, thus activating</u> it;
  - 4. Adenylyl cyclase will catalyze conversion of <u>ATP to cAMP</u>, which binds to and activates <u>protein kinase A (PKA)</u>;

5. Activation of PKA will initiate a sequential <u>activation of kinases</u> resulting in a (phosphorylation cascade) to eventually activate glycogen phosphorylase for breakdown of glycogen;

[Total: 10]

- **2** Fig. 2.1 shows the effect of increasing temperature on the activity of three protein-digesting enzymes:
  - thermitase from thermophilic *Termoactinomyces vulgaris*
  - subtilisin from *Bacillus subtilis*
  - modified subtilisin
- (a) Describe, with reference to Fig. 2.1, the effect of temperature on the rate of protein digestion by thermitase. [3]
  - 1. As temperature increases from <u>10°C to 76°C</u>, the <u>rate of protein digestion</u> by thermitase <u>increases</u> from <u>0 arbitrary units(a.u) to 650 arbitrary units(a.u)</u>;
  - 2. The <u>optimum temperature</u> of the enzyme thermitase is <u>76°C</u> where there is <u>maximum rate of</u> <u>protein digestion</u>;
  - 3. Further increase in temperature from <u>76°C to 90°C</u> will cause <u>the rate of protein digestion</u> <u>decreases sharply</u> from <u>650 a.u to 355 a.u;</u>
- (b) Explain the effect on thermitase of increasing the temperature above 80°C. [3]
  - 1. As the temperature increases <u>above 80°C</u>, there will be <u>greater</u> (intramolecular) <u>vibrations/</u> <u>thermal agitation</u> of the enzyme thermitase;
  - 2. which <u>breaks hydrogen</u>, ionic bonds and other weak interactions that stabilizes the 3D <u>conformation</u> resulting in <u>denaturation</u>\*;
  - 3. The enzyme *active site*\* no longer *complementary in shape*\* and charge to the <u>substrate</u>; and
  - 4. the <u>rate of reaction decreases steeply</u> from a rate of 570 a.u. to 340 a.u.;
- (c) Modified subtilisin is similar to subtilisin, but has had eight of its amino acids replaced with different amino acids.

Describe **and** explain the effect of this modification on the activity of subtilisin. [4] *Describe* 

- Modified subtilisin has a <u>higher rate of protein digestion across all temperatures</u> compared to subtilisin; or
- 2. Modified subtilisin has a <u>wider range of temperatures</u> in can work in, from 10°C to 90°C compared to subtilisin which works from 10°C to 74°C;
- 3. Modified subtilisin has an <u>optimum temperature of 76°C</u> <u>compared to</u> subtilisin which has an optimum temperature of <u>59°C</u>;
- 4. The <u>maximum rate of protein digestion</u> in the modified subtilisin is <u>4 times higher</u> than in subtilisin (i.e. <u>319 a.u for modified subtilisin and 78 a.u for subtilisin</u>);

Explain

5. The replacement of eight amino acids in the modified subtilisin has resulted in a more <u>thermostable</u> enzyme;

- 6. The *<u>R groups</u>\** of these amino acids are able to form <u>stronger bonds</u>, such as strong covalent bonds like <u>disulfide linkages between cysteine residues</u>, which can only be broken at higher temperatures;
- 7. Hence, the <u>conformation of the active site</u> remains <u>stable</u> and is able to catalyse the reaction longer and at higher temperatures;

[Total: 10]

- **3(a)** Define each of the following types of stem cell and name a naturally occurring example of each.
  - (i) totipotent [2]
    - They have the ability to <u>differentiate</u>\* into <u>all cell types</u> that make up an organism including <u>extra-embryonic tissue</u>\* such as <u>placenta</u>\*, and hence are able to form <u>entire organism</u>;
      - 2. e.g. zygotic stem cells/ fertilised egg up to 8 cell stage/first three divisions;
  - (ii) pluripotent [2]
    - They have the ability to <u>differentiate\*</u> into almost <u>all of the cell types</u> that make up an organism <u>except extraembryonic tissue</u>\* such as <u>placenta\*</u> but <u>cannot form the entire</u> <u>organism alone;</u>
    - 2. e.g. inner cell mass of blastocyst\*;
  - (iii) multipotent [2]
    - 1. They have the ability to *differentiate*\* into only <u>a limited and related range of cell types</u> <u>and tissues</u> in an organism to replace dead cells that died;
    - 2. e.g blood/haematopoietic stem cells in bone marrow;
- (b) Induced pluripotent stem cells (iPSCs) are a type of pluripotent stem cell that are produced directly from adult cells. To produce iPSCs, four genes need to be introduced into adult cells. These genes code for transcription factors.

Explain why these transcription factors are necessary for the production of iPSCs from adult cells. [2]

- 1. These transcription factors such as <u>**repressors/activators**</u>\* bind to the <u>silencers/enhancers</u>\*;
- 2. initiate the transcription of inactive genes in the differentiated somatic adult cells;
- 3. and altered the <u>gene expression patterns</u> of the cells such that it was similar to that of <u>embryonic stem cells</u>; resulting in the production of iPSCs

A: upregulate proto oncogene (see below for example)/inhibit tumour suppressor gene.

- 4. One of these transcriptions factors is an activator that binds to the enhancer of a proto oncogene;
- 5. That upregulates transcription leading to increased cell proliferation;
- 6. Example is a protein that promotes cell division e.g. cyclin, growth factors;
- (c) Explain how the use of iPSCs may overcome some of the ethical concerns of using other types of stem cells in medical research and treatment. [2]
  - 1. As iPS cells are not from an embryo,
    - A. there will be no destruction of <u>embryos</u> and hence <u>not regarded as killing a life</u> due to some views that <u>embryos are viable living organisms;</u>

- B. Some object to extracting stem cells from an <u>embryo</u> to make replacement body cells is treating <u>embryo as just a source of spare parts;</u>
- C. Once human status is denied to <u>embryos</u>, this precedent may extend to other categories of human beings such as profoundly <u>disabled or elderly infirm</u>;
- 2. Since the patient's own iPSCs can be used, there will be no <u>foreign tissues</u> or <u>antigens</u> introduced into the patient and thus <u>will not</u> result in <u>immune response/tissue rejection</u> and thus there will be <u>no need for immunosuppressant drugs</u>;

[Total: 10]

- **4** Fig. 4.1 shows the main structural features of the influenza virus.
- (a) With reference to structures shown in Fig. 4.1, explain how an enveloped virus such as influenza:
  - (i) enters a host cell [4]
    - 1. *Haemagglutinin\** binds to *sialic acid receptor\** on host cell membrane;
    - 2. Virus then enters host cell by <u>endocytosis\*</u> where host cell membrane <u>invaginates\*</u> and pinches off, placing virus in an endocytotic <u>vesicle;</u>
    - 3. Vesicle then <u>fuses\*</u> with a <u>lysosome</u>\*, and the resulting drop in pH stimulates <u>fusion of</u> <u>viral envelope with vesicle membrane;</u>
    - 4. Releasing *nucleocapsid*\* and eventually viral RNA into cytosol;
  - (ii) exits a host cell [2]
    - 1. <u>Capsid</u> proteins associate with the <u>ribonucleoprotein</u> (viral genome and proteins) near host plasma membrane;
    - 2. Each new virus <u>buds</u> from the host cell;
    - 3. Acquiring viral <u>envelope</u> from the host plasma membrane which has <u>neuraminidase and</u> <u>haemagglutinin embedded;</u>
    - 4. Neuraminidase cleaves sialic acid to release progeny virions from host cell surface.

Fig. 4.2 shows two examples of antigenic shift in the influenza virus. The resulting human viruses were responsible for the 1957 and 1968 influenza epidemics.

# Fig. 4.2

- (b) With reference to Fig. 4.2, explain how antigenic shift occurs. [2]
  - Antigenic shift occurred where the <u>RNA segments</u> from the <u>2 different influenza virus</u> packaged into the <u>same</u> virion, giving rise to <u>random reassortment</u> of <u>RNA segments</u> resulting in <u>new</u> <u>combination of glycoproteins in the novel virus;</u>
  - 2. Either

In 1957, <u>H2N2 bird virus and H1N1 human virus</u> infects same cell giving rise to <u>new H2N2</u> <u>human virus</u> with new combination of RNA segments;

Or

In 1968, <u>H3 bird virus and H2N2 human virus</u> infects same cell giving rise to <u>new H3N2 human</u> <u>virus</u> with new combination of RNA segments;

(c) Viruses such as influenza rapidly accumulate gene mutations.

Explain the advantage of this to the virus. [2]

- 1. Rapid mutations results in <u>antigens</u> like haemmaglutinin and neuraminidase having a <u>different conformation;</u>
- 2. Thus cannot be recognized by *memory*\* T and B cells of the immune system immune cells;
- 3. So it takes a <u>longer time</u> to <u>remove the virus</u> from the body allowing them opportunity to <u>replicate</u>;

[Total: 10]

- **5 (a)** Describe **two** ways in which the *lac* operon is similar to the *trp* operon. [2]
  - 1. Both consist of several genes under the control of a single promoter
    - 2. Both have an operator region for the binding of an active repressor that prevent transcription
    - 3. The genes code for enzymes involve in the same metabolic pathway or functionally related proteins are synthesised as a unit;
    - 4. Each operon when expressed produce a polycistronic mRNA that has several start codons and stop codons
- (b) Describe four ways in which the *lac* operon is different from the *trp* operon. [4]

	<i>Lac</i> operon	<i>Trp</i> operon		
Type of operon	Inducible operon that is switched on	Repressible operon that is switched		
	in the presence of an inducer	off in the presence of excess end		
	lactose	product tryptophan		
Effector	Inducer is lactose which is the	Corepressor tryptophan, an end		
molecule	substrate	product		
Repressor	Active form	Inactive form		
synthesized in				
Condition	Allolactose binds to and inactivates	Tryptophan binds to and activates the		
affecting	the repressor such that it cannot	repressor such that it can bind to the		
repressor	bind to the operator	operator		
Type of	Enzymes produced involved in a	Enzymes produced involved in an		
metabolic	catabolic process of breaking down	anabolic process of synthesizing		
pathway	lactose	tryptophan		
Default operon	Operon is switched off by default	Operon is switched on by default until		
expression	until the presence of lactose	tryptophan is in excess		
No. of genes/	lac operon codes for 3 different	trp operon codes for 5 different		
proteins coded	enzymes	enzymes		

(c) Suggest the advantages to bacteria of arranging some genes in operons. [4]

1. Operon contain group of genes under the control of the same promoter that allow for <u>functionally</u> related proteins to be synthesised as a unit;

2. An operon can be <u>turned 'on' or 'off' according to certain changes/conditions</u> so as to <u>respond</u> <u>rapidly and appropriately</u> to the environment;

- 3. Bacterium only <u>produces enzymes when required</u> allowing the bacteria to make <u>economical</u> <u>use of energy and resources/conserve resources</u> i.e. relevant genes are expressed only when necessary;
- 4. Especially since bacteria are able to <u>use a variety of metabolites</u> e.g. glucose is metabolised preferentially over lactose, thus not economical to produce *lac* genes in the presence of glucose;
- 5. The above mentioned provide a selective advantage to such bacteria

[Total: 10]

- **6** Fig. 6.1 shows an example of the sequence of temperature changes required for one cycle of a polymerase chain reaction (PCR).
- (a) During the PCR cycle shown in Fig. 6.1, three different temperatures are required.

Explain why each of these **three** different temperatures is required during one PCR cycle. [3] *Note: Quote temperatures from the graph not the ones you memorise!* 

- Heating to <u>95 ℃\*</u> causes the <u>hydrogen bonds</u>\* between complementary bases of each strand to <u>break</u>, causing denaturation of <u>double-stranded DNA into single-stranded DNA</u> and exposes the bases for complementary base pairing;
- Lower temperature of <u>55 ℃</u>\* allows <u>primers\*</u> to <u>anneal</u>\* specifically to the regions flanking the target DNA sequence via <u>complementary base pairing\*</u>, providing a <u>free 3'-OH group</u> for chain extension;
- Subsequent temperature of <u>70 °C</u>\* is the optimum temperature of <u>Tag polymerase</u>\* which performs the chain <u>extension/elongation</u>\* by synthesising the complementary DNA strand from the 3' end of the primer;

# BUT if question was a 6 mark question, please elaborate:

### 95°C

- Heating to 95°C causes the weak <u>hydrogen bonds</u>\* between <u>complementary bases</u> of each strand to <u>break</u> due to increased molecular vibrations;
- 2. The denaturation of <u>double-stranded DNA into single-stranded DNA</u> exposes the bases for <u>complementary base pairing;</u>

### 55°C

- Lower temperature of 55°C allows <u>primers\*</u> to <u>anneal</u>\* <u>specifically</u> to the regions flanking the target DNA sequence via <u>complementary base pairing</u>\*;
- 4. The primers <u>determine the segment to be amplified</u> and provides a <u>free 3'-OH group</u> for chain extension;

# **70°C**

- 5. Is the optimum temperature of <u>*Tag polymerase*</u>\* which performs the <u>synthesis of</u> <u>*complementary*\* DNA</u> strand;
- Chain <u>extension/elongation\*</u> occurs <u>from 3' end</u> of primer which provides <u>free 3' OH</u> required by polymerase;

- (b) Explain the role of primers in the polymerase chain reaction. [2]
  - 1. Primers are short, single stranded DNA sequences that can anneal to the target DNA sequences;
  - 2. they are <u>complementary</u>\* to the <u>regions flanking the gene of interest</u> and thus <u>determine the</u> <u>segment to be amplified;</u>
  - 3. they provide a <u>free 3'-OH group for chain extension</u> by <u>DNA polymerase</u> so that the gene of interest can be amplified;

# Teacher's comment:

Many students did not mention point 1. It is useful to take note that when describing the role, it is good to briefly mention the key structural feature.

For point 2, many students wrote that the primers bind to the 3' ends of single-stranded DNA but this statement is incorrect because a single-stranded DNA strand can comprise several genes but primers bind to the 3' ends of the target gene only.

#### For example, INCORRECT x



CORRECT to specifically amplify Gene 2  $\checkmark$ 



For point 3, most students were able to score the marks for this. However, those who failed to mention that the free 3'OH was needed by the DNA/ Taq polymerase were not awarded the marks.

(c) PCR products are usually separated by gel electrophoresis.

Outline the main principles that allow gel electrophoresis to separate DNA fragments. [3]

- 1. <u>Dense</u> loading buffer is mixed with DNA sample to help it <u>sink</u> to the bottom of <u>wells located</u> <u>nearest the negative electrode/cathode;</u>
- 2. **<u>Negatively-charged\* DNA</u>** migrates out of well towards <u>direction of positive electrode/anode</u> when subjected to an electric field / current;
- 3. Buffers contain ions which allows conduction of electric current;
- 4. <u>Meshwork</u> of agarose polysaccharides <u>impede movement of longer fragments more</u> than shorter fragments;
- 5. causing them to migrate slower than shorter fragments and end up nearer to the well;
- (d) Marker DNA and three DNA samples were separated by gel electrophoresis.

### Fig. 6.2. shows the DNA banding patterns after visualisation.

### Explain why marker DNA was included in this experiment. [2]

- 1. The marker DNA consists of a mixture of DNA fragments of known sizes;
- 2. it acts as a standard that the fragments of unknown sizes from the 3 DNA samples can be compared with to <u>estimate their fragment size;</u>

[Total: 10]

7 Two pure-breeding varieties of summer squash, one producing white fruit and one producing green fruit, were crossed. All the F1 generation progeny produced white fruit.

The F1 plants were then self-pollinated. In the F2 generation, 236 plants produced white fruit, 56 plants produced yellow fruit and 12 plants produced green fruit.

The control of the fruit colour in the summer squash is an example of dominant epistasis. Dominant epistasis is expected to result in a 12:3:1 ratio of offspring phenotypes in the F2 generation.

## (a) Explain the term epistasis in this context. [3]

- 1. Colourless precursor  $\rightarrow$  yellow  $\rightarrow$  green pigment by 2 enzymes.
- 2. Genotype A\_ results in production of <u>inhibitor</u> that <u>prevents colourless precursor</u> to be converted to <u>yellow</u> <u>pigment</u>
- 3. Genotype B\_ results in production of <u>inhibitor</u> that <u>prevents yellow pigment</u> to be converted to <u>green</u> <u>pigment</u>
- 4. <u>Genotype A masks</u> the phenotypic expression of the <u>B/b locus / B genotype</u>.
- 5. Genotype A\_results in white squash since the inhibitor prevents production of any subsequent coloured pigments.

### Alternative Pathway:

White  $\rightarrow$  yellow (in the presence of allele B) White  $\rightarrow$  green (in the presence of bb genotype) Remains as white in the presence of A allele which codes for an inhibitor for enzyme B/b

(b) Draw a genetic diagram to explain the results of crossing the F1 offspring to produce the expected 12:3:1 phenotypic ratio in the F2 generation.

Use the symbols **A**, **a** and **B**, **b** to represent the alleles. [4]

Let A represent the dominant allele for production of inhibitor that prevents colourless precursor to be converted to yellow intermediate. Let a represent the absence of inhibitor.

Let B represent the dominant allele for production of inhibitor that prevents yellow intermediate to be converted to green pigment. Let b represent the absence of inhibitor.

or

Let A be the dominant allele for coloured and a be the recessive allele for white

Let B be the dominant allele for yellow and b be the recessive allele for green

F1 phenotype	White	Х	White

F1 genotype Fertilisation to pro	duce F <sub>2</sub>	AaBb	AaBb	
Gametes	AB	Ab	aB	ab
AB	AABB	AABb	AaBB	AaBb
	white	white	white	white
Ab	AABb	AAbb	AaBb	Aabb
	white	white	white	White
aB	AaBB	AaBb	aaBB	aaBb
	white	white	yellow	Yellow
ab	AaBb	Aabb	aaBb	aabb
	white	white	yellow	green
F <sub>2</sub> genotypes	<b>9 A_B</b>	: <b>3 A_bb</b> ;	3 aaB_:	1 aabb
F2 phenotypic ratio	o <u>1</u>	2 white :	3 yellow :	1 green

(c) A chi-squared test was carried out to compare the expected number of each phenotype in the F2 generation with the observed number. This is shown in Table 7.1.

Explain how the chi-squared test value of 2.88 supports dominant epistasis as the correct explanation of these results. [3]

- 1. Since <u>0.10
- 2. Therefore p > 0.05 at 5% level of significance, we do not reject the null hypothesis that there is no significant difference between the expected and observed phenotypes
- 3. <u>Difference between observed and expected results is not significant</u> and <u>due to chance</u>, therefore supporting dominant epistasis as the correct explanation;

[Total: 10]

8 Different respiratory substrates are available to working muscles to maintain the ATP levels required for muscle contraction. These include glucose and fatty acids from the blood and glycogen in muscle.

Table 8.1 shows the percentage contribution to muscle respiration of each of these respiratory substrates for an athlete, during a long-distance run of 40 km. Results are shown at four different times from the start of the run.

- (a) Describe the changes in the percentage contributions to muscle respiration of fatty acids from blood and glycogen in muscle during this long-distance run, as shown in Table 8.1. [3]
  - 1. The percentage contribution to muscle respiration by <u>fatty acids</u> from blood was <u>37% for the</u> <u>first 90 minutes</u> and started to <u>increase\* significantly to 62% at 240 minutes;</u>

- 2. The percentage contribution to muscle respiration by <u>glycogen</u> in muscle was <u>highest at 36%</u> <u>at 40 minutes but **reduced**\* until it reached 8% at 240 minutes;</u>
- 3. The percentage contribution from fatty acids and glycogen was similar in the first 40 minutes but as the run progresses, the percentage contribution started to <u>diverge with a lower</u> contribution from glycogen and a correspondingly higher contribution from fatty acids; (comparison)
- (b) During the long-distance run, ATP may be generated at times using the type of respiration that normally occurs under anaerobic conditions. However, the athlete cannot use this type of respiration continuously throughout the whole period of the long-distance run.

Suggest why this type of respiration cannot be used continuously by the athlete to generate ATP during the long-distance run. [2]

- 1. Under anaerobic conditions, *lactic acid fermentation*\* occurs;
- 2. <u>Lactic acid accumulates</u> in the muscles resulting in <u>muscle fatigue;</u>
- (c) Explain the different patterns of change in the percentage contributions to muscle respiration of glucose from food and glycogen in muscle during the long-distance run, as shown in Table 8.1. [4]
  - 1. Over the duration of the run, the <u>percentage contribution to muscle respiration from glycogen</u> in muscle <u>decreases over time</u> because glycogen, a <u>carbohydrate store</u> in the muscle is progressively <u>used up</u>; data not required as mark was already allocated in part (a)
  - 2. There was a <u>significant increase</u> to muscle respiration by <u>glucose from food from 27% to 41%</u> <u>at the 90 minute mark</u>; *Quote for glucose*
  - 3. This <u>delay in increase</u> could be a result of <u>time taken to digest the food/absorption of glucose;</u>
  - 4. From the <u>90 minute onwards</u>, the contribution by glucose from food gradually <u>decreases to 30%</u> (as less food is available to generate glucose/fatty acids becomes a major energy contributor);
  - 5. The changes in pattern is dependent on the energy sources available over time. <u>Glycogen is a</u> ready energy source that is used first while glucose from digestion of food is used next;
- (d) Suggest why fatty acids are **not** used more in the earlier stages of the long-distance run. [1] It takes time to convert fat into energy. Lipase will need to breakdown fat to release fatty acids followed by oxidation of fatty acids to give rise to acetyl-CoA;

[Total: 10]

- 9 Fourteen different species of Darwin's finches are found in the Galapagos Islands. These finches have evolved from a common ancestor during the past 1.5 million years.
- (a) With reference to Fig. 9.1, explain why the large ground finch, medium ground finch and small ground finch are considered to be three separate species. [3]
  - The 3 different finches have their <u>own distinct body masses and beak length</u> in their populations. The <u>large ground finch population has body mass between 32 -39g and beak length between 36-42mm</u>, the medium ground finch population has a body mass between 18-24g and beak length <u>between 24-28mm</u> and the small ground finch population has a body mass between 10-15g and <u>beak length between 16-18mm</u>;
  - 2. These <u>phenotypic differences</u> in body mass and beak length are <u>due to differences in their genes</u> and hence they may be <u>genetically different</u> from one another;
  - 3. This could be due to <u>geographical isolation</u> (different islands) hence leading to <u>reproductive isolation</u> such that they <u>no longer can interbreed and form viable offspring</u>, leading to speciation;

(b) Discuss how the information in Fig. 9.1 and Fig. 9.2 shows how these two processes may have contributed to the evolution of Darwin's finches.

### micro-evolution

- 1. Microevolution occurred over a few generations after the ancestral population with <u>variation in beak</u> <u>length arrived;</u>
- As <u>beak lengths</u> <u>best adapted</u> to the <u>different food sources</u> (fig.9.1) were <u>selected for</u> resulting in <u>changes to the allele frequencies within this ancestral species</u>; Hence microevolution occurred

## macro-evolution

- Due to <u>rising sea levels</u>, more islands formed (fig.9.2), and geographical isolation of the ancestral finch sub populations in the occurred that <u>prevented interbreeding</u> and hence <u>disrupted gene flow</u>\*;
- the isolated subpopulations were subjected to <u>genetic drift\*</u> and <u>accumulation of mutations\*</u> and <u>natural selection\*</u> and over <u>many generations</u> evolved independently into <u>new species</u>. Hence macroevolution occurred; [4]
- (c) Fig. 9.3 shows the phylogeny based on whole genome sequencing of the six closely related species of Darwin's finches that have evolved most recently.

Describe the advantages of using genome sequences in reconstructing phylogenetic relationships. [3]

- 1. Nucleotide data are <u>objective</u>. <u>Molecular character states are unambiguous</u> as A, C, G and T are easily recognisable and cannot be confused;
- Nucleotide data are <u>quantitative</u>. Molecular data are <u>easily converted to numerical form</u> and hence are amenable to mathematical and <u>statistical analysis</u> and hence computation. <u>Degree</u> <u>of relatedness can be inferred and quantified</u> by calculating nucleotide differences between species;
- 3. Nucleotide data can be used to <u>compare species which are morphologically indistinguishable</u> especially if they are very closely related like the finches;
- 4. As changes in nucleotide sequences accumulate over time with clockwork regularity. We can estimate the time of speciation of modern to ancient species and <u>place the speciation events</u> on a timescale above;

[Total: 10]

- **10** Fig. 10.1 shows the percentage of the population having the TB vaccination and the rate of TB transmission from 1980 to 2010 in country **P**.
- (a) Describe and explain the changes in the rate of TB transmission that are shown in Fig. 10.1. [3]
  - 1. **Trend**: As percentage TB vaccination increased drastically from 40% to 100% 1980 to 1989, the rate of TB transmission per 100 000 decreased sharply from 8.0 to less than 3.7.
  - Trend: From 1989 to 2010, when percentage TB vaccination remained relatively high above 83%, transmission remained relatively low below 5.0 per 100 000; OR
  - 3. **Trend**: However the <u>inconsistent percentage of TB vaccination</u> from 1989 onwards resulted in a <u>base/minimum rate of transmission of at least 3.4 per 100 000</u>
  - 4. **Explanation**: as a <u>larger percentage</u> of the children are <u>vaccinated</u>, <u>more children were immune</u> and protected against TB and <u>did not contribute to the transmission</u> of the disease.

5. Quote any complete set of data from both axes

Teacher's comment:

- Many students did not use descriptive words for the shape of the graphs such as sharp or drastic decrease and steep increase
- Students should observe the trend for both graphs is from 1980 to about 1989 and from 1989 to 2010 as the shape of the graph decrease/increase sharply then fluctuates around a certain value. With only 3 marks should not describe every part of the graph. They should also ignore the peak at 1987.
- When reading from a graph with 2 axes from both sides, students need to be more careful. Some students read the axis wrongly in % rather than rate or the rate value wrongly e.g 350000 instead of 3.5 per 100000.
- Many students mistaken the graph represents herd immunity. For herd immunity, the transmission rate is much lower. They should take the cue from Fig. 3.2 that Q shows herd immunity thus Fig. 3.1 where the vaccination fluctuates and goes below 95% with higher rate of transmission does not.
- (b) Fig. 10.2 shows the percentage of the population having the TB vaccination and the rate of TB transmission from 1980 to 2010 in country **Q**.

Suggest reasons for the differences in the rate of TB transmission between country P and country Q, as shown in Fig. 10.1 and Fig. 10.2. [2]

Either 1 or 2 for 1 mark

- Country P had a rate of transmission that is erratic and <u>varied between 3.3 and 5.0 per 100 000</u> from <u>1989</u> while country Q has a <u>consistent drop in rate of transmission from 4.5 to 2.5 per 100</u> <u>000</u> from 1989;
- <u>Country Q had a more consistent/robust/compulsory vaccination programme</u> that ensured that <u>98% of the children were immunised from 1989</u> while <u>country P could not maintain a high</u> <u>percentage that at times fell to 83%; - reason 1</u>

Either 3 or 4 for 1 mark

- 3. This meant that country Q experienced *herd immunity*\*; reason 2
- 4. <u>With a sufficiently large proportion of immunised individuals (of above 95%) who cannot spread</u> <u>the disease</u>, the <u>entire community</u> including the individuals who are <u>not vaccinated/cannot be</u> <u>vaccinated are protected because transmission is prevented</u>; - *explanation of herd immunity*

note: the unvaccinated are still vulnerable

Teacher's comment:

- As this is a comparison question, students must state data clearly from both countries Q and P. A few students mentioned wrongly comparing Q vs Q instead of Q vs P.
- As herd immunity is not established in P, Q should not refer to as showing stronger herd immunity.
- In explanation for pt 4 most students are not able to explain clearly.

[Total: 5]

- **11** Fig. 11.1 shows the concentration of dengue virus antigen and antibody in the blood of a person following infection with the virus for the first time.
- (a) Describe how the concentrations of dengue virus antigen and antibody change during the first ten days following infection, as shown in Fig. 11.1. [2]
  - 1. As the dengue virus <u>antigen</u> concentration <u>increases steeply</u> from <u>Day 1</u> to <u>peak</u> at <u>Day 3</u>, the <u>antibody</u> concentration <u>gradually increases</u>;
  - 2. As the antibody concentration increases <u>steeply/exponentially</u> from Day 3 to <u>plateau</u> at <u>Day 10</u>, the virus antigen concentration <u>decreases steadily</u> from <u>Day 3</u> to <u>zero</u> at <u>Day 9</u> and 10;
- (b) Outline the role of antibodies in eliminating the dengue virus from the blood. [3]
  - Two antigen binding sites per antibody molecule allows <u>agglutination</u>\* of the pathogen, able to bind to two antigens/pathogens at the same time → pathogens <u>aggregate/clump</u> to facilitate clearance
  - 2. <u>Neutralisation</u>\* of the pathogen by the antibody which <u>prevents the entry</u> of pathogen/toxins into the host cells
  - Fc region/constant region of heavy chains bind to Fc receptors on phagocytes to tag / promote phagocytosis which is opsonisation\*;

[Total: 5]