VICTORIA JUNIOR COLLEGE
BIOLOGY DEPARTMENT
JC2 PRELIMINARY EXAMINATIONS 2016
Higher 2

BIOLOGY 9648/02

Paper 2 Core Paper Answers **14 September 2016**

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1 (a)
(i) Identify structures **P** and **Q** and describe briefing their functions. [3]

- P – Nucleolus [1/2]
- Transcription of ribosomal RNA [1/2]
- Site of ribosome assembly [1/2]
- Q – Smooth endoplasmic reticulum [1/2]
- Site of synthesis of lipids [1/2]
- Detoxification of drugs and poisons [1/2]
- Stores calcium ions required for contraction in muscle cells [1/2]

(ii) Contrast the structure of a lysosome with structure **P**. [2]

Lysosome	P (Nucleolus)
• Membrane-bound [1/2]	• Not membrane-bound [1/2]
• Contains hydrolytic enzymes [1/2]	• Contains DNA coding for rRNA [1/2]

(b)
(i) Name a carbohydrate that functions as a storage molecule for T-helper cells.

- Glycogen [1]

(ii) Describe **three** structural differences between cellulose and the carbohydrate in **(bi)**. [3]

Cellulose	Glycogen
• Made up of β-glucose	• Made up of α-glucose
• Joined by β 1,4 glycosidic bonds	• Joined by α 1,4 glycosidic bonds and α 1,6 glycosidic bonds
• Unbranched, straight chains	• Branched brush-shaped
• Alternate subunits rotated 180^{o}	• Alternate subunits in the same orientation
• Inter-chain hydrogen bonds present	• No cross-linkages between adjacent chains

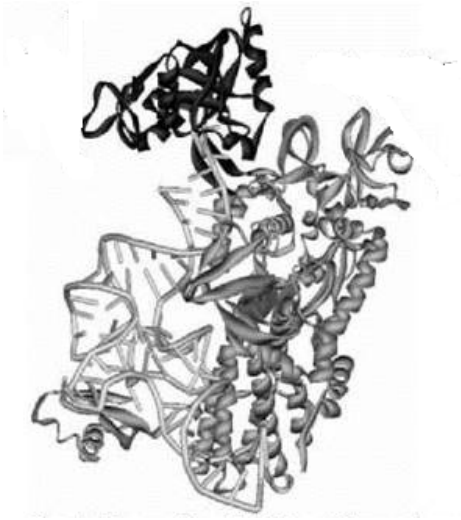
(iii) Explain how the presence of two types of bonds in amylopectin enables it to carry out its function. [2]

- α 1,4 glycosidic bonds between subunits within a branch [1/2]
- α 1,6 glycosidic bonds at branch points [1/2]
- form branched helical structure
- compact for storage function
- Both bonds can be broken enzymatically to release α glucose for respiration [1/2]
- Hydrolysis of α 1,6 glycosidic bonds breaks up amylopectin into many branches for more efficient breakdown [1/2]

(c) Phosphofructokinase is an allosteric enzyme. Explain how the presence of an allosteric inhibitor affects the enzymatic activity of an allosteric enzyme. [2]

- Allosteric inhibitor binds to allosteric site [1/2]
- Causes the enzyme conformation to change to inactive state [1/2]
- Active site not complementary to substrate [1/2]
- Prevent effective collision and formation of enzyme-substrate complex [1/2]

2. The diagram below shows an enzyme involved in the activation of tRNA for translation in prokaryotes.



(a) (i) Explain the mode of action of this enzyme [3]

- amino-acyl tRNA synthetase;
- has a specific active site that is
- complementary to specific tRNA anticodons and a specific amino acid
- Ref. to induced fit theory
- catalyses the attachment of a specific amino acid to the 3' stem of the tRNA in the formation of the amino-acyl tRNA complex
- by lowering the activation energy of the reaction
- through the formation of an enzyme structure complex

(ii) Explain the significance of having more than one type of the enzyme named in (ai) in the cell [2]

- There are 20 different amino acids and hence 20 different amino-acy-tRNA synthethases are needed
- This ensures that each of the 20 amino acids are correctly linked to their tRNAs/ ref to specificity of enzyme for substrate
- As the anticodons of the tRNA bind by complementary base pairing to the condons in the P and A site of the ribosome
- When an amino acid has been linked to a tRNA, it will be incorporated into a growing polypeptide chain at a position dictated by the codon of the mRNA.
- Allowing the primary structure of the polypeptide to be synthesised correctly according to the codons of the mRNA that is being translated

(b) How does the order of nucleotides in a gene encode the information that specifies the primary structure of a polypeptide? Include two features of the genetic code in your answer.[3]

- Transcription of the gene by RNA polymerase produces a complementary sequence of mRNA;
- Three consecutive nucleotides on mRNA make one codon;
- One codon codes for one amino acid;
- Although more than one codon can code for the same amino acid due to the degenerate nature of the genetic code;
- The ribosome read the codons one after another with no space between codons as the genetic code is non-overlapping.
- The ribosome thus joins the amino acids in the correct sequence as coded for by the codon sequence to form the polypeptide's primary structure/ the codon sequences hence determine the number, type and sequence of amino acids of the polypeptide synthesized by the ribosome
- As the genetic code is punctuated where 3 codons do not code for amino acids but function as stop codons that mark the end of translation. The ribosome stops polypeptide synthesis when a stop codon is located in the ribosome A site as a release factor enters the site to release the completed polypeptide.

(c) Explain how different polypeptides can be synthesised simultaneously from a single mRNA in prokaryotes. [2]

- A prokaryotic mRNA is a polycistronic mRNA;
- And contains the coding sequence for more than one polypeptide/ structural gene product involved in a related metabolic pathway;
- Each coding sequence has its own start and stop codon;
- Allows more than one ribosome to bind to the polycistronic mRNA and start simultaneous translation beginning at the start codon
- more than one translation initiation complex can be formed at a time;
- Translation of each polypeptide stops when the ribosomes read the stop codon for the coding sequence

3

(a) Using Fig 3.1, explain the mode of control of the Arg operon. [2]

- Negative control of arg operon;
- as the repressor (activated by arginine) is required to switch / turn off gene expression;

(b) Explain why it is useful for a bacterial cell to decrease expression of the structural genes when arginine is present. [2]

- Trp genes code for enzymes (involved in / necessary for) (anabolism / synthesis) of tryptophan
- Decreased expression helps to conserve resources that could be diverted for other uses /preventing wastage of resources

(c) Name and describe the process which can result in a population of bacteria acquiring the same allele needed to increase their likelihood of survival.

- **specialised transduction;;**
- Viral DNA **integrates** into a **specific location**;
- When it excises as the cell enters the **lytic cycle**;
- The bacterial DNA removed along with the **excision** of the viral DNA;
- will be those that are **near to the prophage** on the bacterial chromosome;
- DNA transferred will therefore be about the same;

(d)(i) Briefly describe the role of DNAase in this experiment.

- Digest naked DNA fragments

(ii) How does the lack of DNAase in the experiment result in the growth of the hybrid bacterial colonies?

- Without the DNAase, the naked DNA fragments from bacteria which have died may be taken up by the other strain via transformation
- DNA fragment will be small enough to cross over the filter

4.

With reference to Fig. 4.1

(a) (i) describe the effect of the 2 gene mutations on the occurrence of cancer in Asians. [1]

- 70% of never smokers with lung cancer have mutations in EGFR gene compared to 27% of ever smokers with lung cancer;
- While 4 % of never smokers with lung cancer have mutations in Ras gene compared to 20% of ever smokers with lung cancer;

(ii) suggest how never smokers in Asia developed lung cancer [2]

- never smokers who get lung cancer could have inherited a dominant mutation in the EGFR gene and experienced a loss of heterozygosity for two or more tumour suppressor genes;;
- as seen from the high percentage of never smokers having the mutation in the EGFR gene compared to ever smokers suggesting the never smokers inherited an increased disposition to acquiring lung cancer;
- even in the absence of exposure to chemical carcinogens such as tar in cigarette smoke

(b) Suggest the role of the *EGFR* gene in relation to the development of cancer. [2]

- *EGFR* gene is a proto-oncogene;
- That codes for the production of growth factor receptor involved in the signalling pathway for cell division;
- Gain-of-function mutation converts EGFR gene into an oncogene;
- The hyperactive/ constitutively dimerised EGFR protein/ receptor constantly stimulates cell division/ results in abnormally active signalling to initiate cell division
- The cell is able to proliferate in the absence of growth factors
- The excessive/ uncontrolled cell proliferation leads to formation of cancerous tissue / tumor.

(c) Small molecule inhibitors of the tyrosine kinase enzymatic activity to inhibit autophosphorylation and signalling of the EGFR have been used in clinical treatment in the United States. Results found that the success of treatment of lung cancers is higher for never-smokers than ever-smokers. Based on Fig. 4.111, suggest why this is so. [2]

- Mutations in EGFR is causal to development of cancer in 40% of never-smokers compared to 10% of ever-smokers;
- Hence inhibiting the EGFR is 4 times more likely to lead to success in treatment in never-smokers;
- A higher percentage of ever-smokers (22% compared to 14%) have mutations in Ras;
- Ras acts downstream of the EGFR / involved in another cell signalling pathway;
- A EGFR inhibitor will have no effect as a hyperactive Ras can signal of excessive cell division even in the absence of signalling from the growth factor receptor;

(d) 50% of never smokers with lung cancer also have mutations in the p53 gene. Explain how mutations in the *p53* gene may lead to the development of lung cancer. [3]

- p53 is a tumor suppressor gene that serves to restraint cell division;
- ref. Loss-of-function mutations
- ref. in both alleles of the *p53* gene
- ref. p53 protein is a transcription factor
- When there is DNA damage,the mutated p53 protein is unable to activate:
  - DNA repair genes to repair the DNA damage
  - P21 gene stop the cell cycle to allow time to repair DNA
  - genes controlling apoptosis that cause the (lung epithelial) cell to die when there is excessive DNA damage
- thus allowing accumulation of mutations to occur.

5.

(a) (i) State the genotype and phenotype of the clover plant that was self-pollinated.

Genotype: AaBb (1/2)

Phenotype: Normal petals **(1/2)** [1]

(a) (ii) Use a genetic diagram to illustrate the phenotypic ratio of the offspring from this cross.

Phenotype of parents: **normal petals** **x** **normal petals**

Genotypes of Parents: **AaBb** **x** **AaBb**

F1 Gametes **AB** **Ab** **aB** **ab** ; **1 m for gametes;**

F1 Genotypes and Phenotypes:

Gametes	AB	Ab	aB	ab
AB	AABB normal	AABb normal	AaBB normal	AaBb normal
Ab	AABb normal	AAbb fused	AaBb normal	Aabb fused
aB	AaBB normal	AaBb normal	aaBB small	aaBb small

ab	AaBb normal	Aabb fused	aaBb small	aabb fused

1 m for all correct F1 genotypes;

1 m for all correct

F1 Phenotypic Ratios: normal : small : fused

9 : 3 : 4 ; **1 m for correct ratio;** [3]

(b) (i) State the <u>expected</u> phenotypic ratio of the offspring in Table 5.1.

1:1:1:1 [1]

(ii) A X^2 (chi-squared) test was conducted on the results. Using the formula and table of probabilities given below, calculate the X^2 value and give the conclusion that may be drawn from it.

$$\chi^2 = \sum \frac{(\text{Observed Value} - \text{Expected Value})^2}{(\text{Expected Value})}$$

Calculated X^2 value = 67.36 **[1/2]**

Degree of freedom = 3

Probability = < 0.001

Distribution of X^2

	probability, p				
degrees of freedom	**0.10**	**0.05**	**0.02**	**0.01**	**0.001**
1	2.71	3.84	5.41	6.64	10.83
2	4.61	5.99	7.82	9.21	13.82
3	6.25	7.82	9.84	11.35	16.27
4	7.78	9.49	11.67	13.28	18.47

Conclusion:
Since p<0.001

Difference between expected and observed phenotypic ratio is significant and not due to chance;
Observed ratio does not follow expected ratio of 1:1:1: [2]

(b) (iii) Explain the observed phenotypic ratio in Table 5.1.

- Linked genes
- Allele **T** is linked to allele **Y** and allele **t** is linked to allele **y**;
- If crossing over occurs, linkage between alleles is broken / new allele linkages will be formed;
- Such that recombinant gametes/chromosomes (**Ty** and **tY**) are formed ;
- Large no. of tall, yellow-flowered and dwarf, white-flowered / offspring with parental phenotypes OR small no. of dwarf, yellow-flowered and tall, white-flowered / offspring with recombinants phenotypes; [Max 2]

6 **(a)** Myasthenia gravis is a disease of the neuromuscular junctions which causes muscular weakness. It develops because the muscle's response to repeated nerve signals declines with time, and the muscles become weak and tired.

(i) State one similarity in the structure of a normal and myasthenic neuromuscular junction as seen in Fig. 6.1a and 6.1b and explain how it aids in synaptic transmission. [2]

Any one:

- Pre-synaptic neurones are able to secrete acetylcholine. [1/2]
- Acetylcholine diffuses across the synaptic cleft [1/2] and bind to the acetylcholine receptors [1/2] present on the post-synaptic membrane [1/2] / muscle cell → membrane depolarisation [1/2]

Or

- Acetylcholine receptors [1/2] present on the post-synaptic membrane [1/2] / muscle cell.
- Upon binding with acetylcholine, ligand / chemical / Na^+-gated channel opens [1/2] → influx of Na^+ [1/2]→ membrane depolarisation [1/2]

(ii) State one difference in the structure as seen in Fig. 6.1a and 6.1b and explain how it affects synaptic transmission. [2]

Any one:

- Unlike normal muscle cell which is deeply folded [1/2], Myasthenic neuromuscular junction has less shallow in-folding [1/2] of the post-synaptic membrane / muscle cell
- Affects the number of acetylcholine receptor [1/2] (or AChR) embedded on the membrane available to bind to acetylcholine

- Abnormal acetylcholine receptor (or fewer normal acetylcholine receptors present) [1/2] present on the post-synaptic membrane / muscle cell
- Cannot bind to acetylcholine [1/2] / bind to auto-antibodies

- Hence, acetylcholine cannot bind [1/2]
- No post-synaptic depolarisation possible [1/2] → no action potential

(b) Describe the mechanism that ensures unidirectional movement of nerve impulses along the axon of a neurone and no overstimulation of the neurone. [2]

- Refractory period [1/2] – short time immediately after an action potential in which the neurone cannot respond to another stimulus [1/2]

<u>Absolute refractory period</u>

- Voltage-gated Na^+ channels are either already opened (during depolarisation phase) or are inactivated (during repolarisation phase) [1/2]
- Cannot initiate an action potential no matter how strong is the stimulus [1/2]

<u>Relative refractory period</u>

- Voltage-gated K^+ channels are open and membrane is hyperpolarised / during hyperpolarisation phase) [1/2]
- An action potential can only be initiated if the stimulus is stronger than usual [1/2]

- Correct mention of both absolute and relative refractory period [1/2]

(c) The diagram shows the action potentials generated over a fixed time, X.

In the space provided, draw the action potentials generated over the same time period if the stimulus is more intense. [1]

- more AP within period X

7

(a) State the type of receptor that glucagon binds to and explain how this receptor is the fully activated. [2]

- **G-protein linked/coupled receptor**
- Binding of glucagon to complementary binding site on receptor cause a **change in conformation** of the receptor which can now bind G-protein

(b) With reference to fig 7.1, briefly explain how cAMP can lead to an increase in blood glucose concentration. [3]

- cAMP is a **second messenger** in the **signal transduction pathway** of glucagon
- cAMP triggers different signal pathways leading to **different cellular responses** such as glycogenolysis and gluconeogenesis
- cAMP activates the **protein kinase A** which phosphorylates and activates the enzymes needed for the glycogenolysis/the breakdown of glycogen to glucose-1-phosphate and glucose
- Gluconeogenesis which is stimulated results in the generation of glucose from non-carbohydrate carbon substrates such as pyruvate and lactate in the liver.
- Inhibition of the enzymes (e.g. glycogen synthase) needed for the formation of glycogen from glucose
- The glucose is then released by facilitated diffusion through glucose carriers on the surface of the liver cells to raise blood glucose concentration

8 (i) Explain why the evolution of *Jadera haematoloma* after the introduction of a non-native soapberry plant in the 1920s is not considered a form of divergent evolution. [1]

- It did not involve the an inherited characteristics / homologous structure (e.g. sharp beak) undergoing modification to perform different functions [1/2]
- nor did it involve speciation to become two or more different species [1/2]
  or
- There is actually a decrease in phenotypic variation [1/2]
- Q.V [1/2]

(ii) With reference to the information and Fig. 7.2, account for the beak lengths over time. [4]

<u>Description of trend</u>

- Variation of beak lengths prior 1920 / before introduction of the non-native soapberry plant / prior 1920 [1/2]
- QV: 5.5 to 9.1mm [1/2]
- Reduction of beak lengths from 1920 onwards / after introduction of non-native plant [1/2]
- QV: 5.5 to 6.5mm [1/2]

Explanation

- Thinner skin of the non-native fruit provides a selective pressure favouring shorter beak / shorter beak bugs are selected for [1/2]

- Possible reason – same access to nutrients / food as the longer beak bugs but able to survive longer than the longer beak bugs since they do not need to channel additional resources to develop a longer beak [1/2]

- Higher reproductive success [1/2]
- pass favourable alleles to the offspring [1/2]
- Higher proportional of alleles coding for shorter beaks in the gene pool over time [1/2]

(ci) Suggest a likely explanation for the results seen in island Y.

- Ref to genetic drift affected the outcome [1/2]

- Since only 40 individuals (or idea of small population) [1/2] were initially taken to the laboratory, by random chance none of the individual carried the alleles coding for the longer beak. [1/2]

Also accept: By random chance, the individuals carrying the alleles coding for the longer beak in the initial population failed to reproduce successfully after one or few generations in the laboratory and so the alleles were removed in the gene pool [1/2]

- Ref to alleles coding for short beak were fixed in the gene pool [1/2]

(ii) Explain the results in Fig 8.4. [2]

- Bug population in island Y suffered from a low variation of alleles for beak length / only have alleles for short beak length [1/2]
- No natural selection [1/2]
- When only thick-skinned fruits were available, the short beaks could not penetrate the skin to obtain the nutrients so entire population became extinct [1/2]

- Bug population in island X had a greater variety of alleles for beak length [1/2]
- When only thick-skinned fruits were available, the bugs with longer beaks could penetrate the skin to obtain the nutrients and are favoured by natural selection and so increase in number over time [1/2]

(c) Analysis of the DNA sequences of the soapberry bugs in both islands before the viral invasion revealed differences that could not be explained by the theory of natural selection alone. How may the neutral theory of molecular evolution and the genetic code account for the differences? [2]

- Neutral mutations [1/2] occurred but did not affect the fitness / reproductive success of the individuals [1/2]

- Mutations occurred in the non-coding regions which comprise a large proportion of the genome did not affect the phenotype [1/2]

- Mutations occurred in the coding regions could still give rise to the same phenotype because:

  - different codons may code for the same amino acid / degenerate nature of the code [1/2]
  - different codon codes for a different amino acid but retained similar property as the amino acid coded by the original codon [1/2]

2016 H2 BIOLOGY PRELIM PAPER 2 ESQ answers

9 (a) Explain how molecular and anatomical homology supports Darwin's theory of natural selection. [6]

Darwin's theory of natural selection

- It is based on **descent with modifications** [1/2] where different species are **related by descent / share common ancestors** [1/2]

- Heritable traits underwent modifications which resulted in the **differences between present species and the ancestral species** [1/2]

Anatomical homology

- Different but related species would share a basic anatomical plan which they inherited from their common ancestor.

- Named example 1: **Pentadactyl limb** [1/2]
- The **limbs** of all mammals / air-breathing vertebrates share a **basic bone arrangement plan** [1/2]
- Modified differently amongst the descendent species → **locomotion** [1/2]
- As adaptation to the **particular environment / selective pressure** [1/2]
- At least **2 examples** [1/2]: Human forelimbs – manipulation; whales – swimming; bats – flying; AVP
- Named example 2: **Vestigial structure** [1/2]
- **Reduced and may be non-functional** in some species but **functional in other species** [1/2]
- Degeneration of structure due to the **absence of selective pressure** which used to be present [1/2]
- At least **1 example** [1/2]: Limbs in snakes – no longer needed / beneficial for locomotion; Hind-limbs in whales – not needed for swimming / ancestor was a land mammal; AVP

Molecular homology

- Similarities in **genetic language of DNA and RNA** [1/2]
- **Universal genetic code** [1/2] among all species

- **Similarities in DNA / RNA / amino acid sequences** in **homologous genes or proteins** [1/2]
- Known **closely related species or members of the same species** sharing a more recent common ancestor have **greater similarity** than **less related species** [1/2]

10b) Explain the significance of mutations and genetic drift in the neutral theory of molecular evolution [6]

- Evolutionary change at the molecular level occurs primarily through neutral mutations [1/2] which do not affect the phenotype of the organism [1/2]
- According to the theory, most of the genetic variation in populations is the result of mutation and genetic drift and not selection [1/2]
- Mutations result in the creation of new alleles [1/2]
- May or may not affect the biological fitness of the individual [1/2]
- However, many mutations which are neutral
- Reasons:
    - Mutations occurred in the non-coding region of the genome [1/2]
- since non-coding sequences make up the majority of the eukaryotic genome [1/2]
    - Mutations in the coding region may not affect the phenotype
- due to the degenerate code where one amino acid may be coded by more than one codon [1/2]
- If mutations in the coding region result in a slightly different protein, it may also not affect the fitness of the individual (i.e. neither detrimental or adaptative) [1/2]

- In subsequent generations, the frequency of the neutral alleles changes due to genetic drift [1/2] which is the random change of allele frequencies in the gene pool of a (especially small) population from one generation to the next [1/2] Through genetic drift, some of the neutral alleles may be over or under represented [1/2] or lost or become fixed [1/2] in the gene pool. The evolutionary change in the population or species is more pronounced if it is small [1/2]
- Ref to small size due to Founder Effect or Bottleneck Effect [1/2]
- Founder effect – when a few individuals become isolated from a larger population and establishes a new population [1/2]
- Bottleneck effect – when sudden change in environment (e.g. any named natural disaster or over-hunting/over-predation) led to drastic reduction in population size [1/2]
- Hence, only neutral mutations and genetic drift are significant to the theory [1/2]

9b)Describe how the response of muscle cells to insulin with respect to cell signalling. [8]

- Insulin is released from the **β cells of the islets of Langerhans** in the pancreas;

- in response to a rise in blood glucose level **above the norm** of 100mg /100cm^3 blood;
- Before the insulin (ligand) binds, the insulin receptors exist as two individual polypeptides **subunits**;
- The insulin is carried by the blood and **binds to insulin receptor** on the target cell to form a hormone-receptor complex;
- The ligand binding causes two receptor subunits to associate closely with each other forming a dimer (ref . to **dimerization**);
- Dimerization **activates the tyrosine kinase** region of each polypeptide;
- Each tyrosine kinase adds a phosphate from an ATP molecule to a tyrosine on the tail of the other polypeptide (ref. to **cross phosphorylation**);
- The fully-activated receptor protein is now recognized by specific **relay proteins** inside the cell;
- Each relay protein will bind to specific phosphorylated tyrosine residues and will undergo a resultant conformation change that activates it;
- Each activated relay proteins triggers a **specific transduction pathway**;
- leading to a **cellular response**;
- As a result of the activation of **different** relay molecules by **one** activated RTK, ligand binding to a RTK may results in **multiple transduction pathways and cellular responses** (see examples below);
- Ref. to **phosphorylation cascade**, in which a series of different proteins in a pathway are phosphorylated sequentially;
- Ref. to **signal amplification** as one ligand can result in many different downstream proteins being activated
- These cellular responses help bring blood glucose levels back to the norm of 100 mg/100 ml blood. Once the norm level is reached, the **negative feedback mechanism** prevents the further release of insulin from the β cells.

Examples of cellular responses:

- Insulin facilitates the transport of glucose into cells by **increasing the number** of **glucose carriers** at the membranes of the cells;
- Upon activation of the insulin receptor, a signal transduction pathway is activated that causes vesicles in the cytoplasm that contain glucose carrier proteins to **move and fuse to the cell membrane**;
- Increase in glucose carrier proteins, **increase in facilitated diffusion of glucose** into cells;
- Stimulate **glycogenesis** - Activates enzymes involved in glycogen synthesis e.g. glycogen synthase which polymerises glucose-1- phosphate (formed from G6P) to glycogen;

9c) Define control elements and explain how they interact with other factors to influence transcription. [6]

Control elements

- Non-coding regions of the genome that function as binding sites of transcription factors [1/2]
- To control the rate of transcription of genes [1/2]
- Include promoters, enhancers and silencers [1/2]

Promoter

- Proximally upstream of the gene it controls [1/2]
- Contains TATA box [1/2]
- Recognised and bound by general transcription factors [1/2]
- Which recruit RNA polymerase to form transcription initiation complex in order to turn on transcription [1/2]

Enhancer

- Recognised and bound by activators [1/2]
- DNA bending protein causes DNA to bend, bringing the bound activator close to the promoter [1/2]
- Bound activator interacts with the transcription initiation complex to increase transcription [1/2]

Silencer

- Recognised and bound by repressors [1/2]
- Bound repressor interacts with the transcription initiation complex to decrease transcription, prevent activator binding or function, as well as causing DNA to be tightly coiled [1/2]

- Ref. to enhancers and silencers being distal to genes they control [1/2]
- Ref. to transcription factors being able to bind to the control elements when DNA is less tightly coiled / in euchromatin form [1/2]
- Due to histone acetylation and lack of DNA methylation [1/2]

10 (a) Explain how recessive alleles may be preserved in a natural population. [6]

D1. Diploidy – eukaryotes are diploid / have two copies of alleles present for each locus; (each gene can have more than 2 alleles but at any one time, a diploid organism can only have 2.)

D2. Recessive alleles carried by heterozygotes; not subjected to natural selection (hidden from selective effect);

D3. Heterozygotes express dominant trait;

D4. Through masking of recessive allele by the dominant allele of the gene;

D5. (Ref. to effect of natural selection) dominant trait selected for;

D6. Variation only exposed to selection in rare occasion when both parents carry recessive allele, and both copies ending in the same zygote (recessive trait selected against);

D7. Recessive alleles being passed on to the offspring when heterozygotes propagate;

B1. Balancing selection;

B2. When natural selection maintains 2 or more forms in population; (Note: each form is a result of a particular genotype i.e. combination of alleles)

B3. Heterozygote advantage;

B4. When heterozygotes have greater fitness / selective advantage over both kinds of homozygotes;

B5. The recessive allele (in heterozygotes) will be maintained by natural selection / natural selection favours recessive alleles;

B6. Frequency-dependent selection;

B7. When fitness of a phenotype declines if it becomes too common in the population;

B8. The different alleles (including recessive alleles) can be maintained within the population; or frequency of different alleles oscillates over time;

N1. Neutral variation;

N2. Mutation resulting in recessive allele ultimately has no effect on survival or reproductive fitness of individual; not selected against/ selectively neutral;

10 (b) Explain the advantages and significance of having a cell signalling system. [6]

- Cell signalling system comprise of 3 stages: signal reception, signal transduction and cellular response;
- Helps ensure crucial activities/reactions occur in the right cells, at the right time and in proper coordination with other cells of the organism;;
- (At signal reception stage) Specific cells detect specific ligands/signalling molecules; (e.g. hormone glucagon binds to receptor on alpha-cells of islets of Langerhans)

- Ligand shape is complementary to receptor found on/within target cell;
- (Signal transduction stage) Ligand binding causes conformation of receptor protein to change; triggering transduction/signal transduction/multi-step pathway; that involves a sequence of changes in a series of different molecules; such as relay proteins and second messengers (e.g. glucagon biding to GCPR causes it to underdo conformational change, in turn activating G protein, which in turn activates adenylyl cyclase, causing increased production of cAMP (second messenger), which in turn activates protein kinase, and triggers phosphorylation cascade)
- (Cellular response stage) Specific cellular response elicited;

Advantages of multistep pathway:

- Signal amplification; some of the molecules in the pathway transmit the signal to numerous molecules at the next step of the series resulting in a large number of activated molecules at the end of the pathway; (e.g. phosphorylation cascade mentioned above)
- Regulation allows for fine tuning and control of cellular response;
- Numerous cellular responses can be elicited from a single ligand molecule; (e.g. activation of numerous transcription factors and consequently genes, like glycogen phosphorylase and glycogen synthase, which catalyse various reactions that ultimately bring blood glucose levels up to the norm)

10c) Describe binary fission and explain how it differs from bacterial conjugation [8]

- First the circular DNA attaches itself to the cell membrane;
- Duplication starts at the origin of replication; and occurs bidirectionally;
- DNA replicates semi-conservatively;
- When the cell divides, the duplicated DNA is separated and the cell membrane folds inwards to form a double layer across the long axis of the cell.
- New cell wall layers are secreted within the membrane layers.
- This divides the cell into two smaller, identical cells;

	Binary fission	Bacterial conjugation
Purpose of process	Form of asexual reproduction to form genetically identical offspring	Way in which bacteria acquire new genetic material
Number of bacterium involved	Involves one parent bacterium only	Involves 2 bacteria (one F+ /donor cell and one F- /recipient cell)
Type of genetic material replicated	The bacterial chromosomes (and plasmids) of the cell is	The F plasmid is copied and transferred to the recipient

and how it is transferred to recipient (conjugation) or progeny (binary fission)	copied and the cell divides to allow each cell to have one copy.	using a cytoplasmic bridge.
Change in size of bacteria/Fate of bacteria after the process	The parent cell enlarges before dividing equally into two/ Parent cell becomes 2 daughter cells	No change in size of the bacteria involved
Length of time needed for process	Takes a longer time as all genetic material and cellular structures like ribosomes have to be duplicated, as well as the synthesis of a new cell wall to divide the bacterium into two	Takes a shorter time as only time needed for the construction of a cytoplasmic bridge and the transferring of the small F plasmid
What is synthesised in the process	Involves duplication of all genetic material, bacterial ribosomes and new cell wall layers	Involves the synthesis of a cytoplasmic bridge for transfer of the F factor from donor to recipient